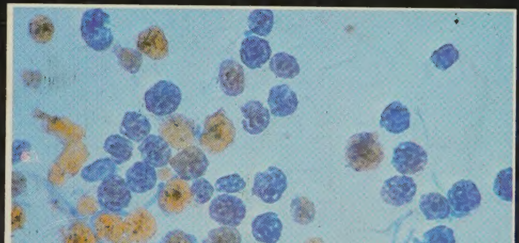
Color Atlas of
POST-HARVEST
Diseases & Disorders of Fruits & Vegetables
Volume 2: Vegetables

Anna L. Snowdon



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Color Atlas of
Post-Harvest Diseases and Disorders of Fruits and Vegetables

Volume 2: Vegetables

Anna L. Snowdon Ph.D., D.I.C.
University of Cambridge

CRC Press
Boca Raton Ann Arbor Boston

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Published in the USA and its Dependencies
by CRC Press Inc., 2000 Corporate Blvd, NW.
Boca Raton, Florida 33431, USA, by arrangement
with Wolfe Publishing Ltd.

Printed by BPCC Hazell Books, Aylesbury, England

ISBN 0-8493-7734-X

Library of Congress Catalog Number has been applied for.

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Preface

In the preface to Volume 1 it is explained how for several years there was a close relationship between the University of Cambridge (Department of Applied Biology) and the UK fruit trade. Specifically, I acknowledge the part played by the National Federation of Fruit & Potato Trades in financing a long-term research and advisory post. This enabled me to carry out extensive surveys of fruit and vegetable consignments, usually in connection with legal claims arising from losses. I am especially grateful to my former research supervisor, Dr Peter Lowings, for introducing me to this world of 'forensic plant pathology'. I thank all the importers who commissioned me to survey their cargoes, and I am indebted to numerous individuals who gave of their time and expertise in the markets, ports and packing stations, both in the UK and abroad.

The book has been designed to be used in two ways. The photographs and text provide the basis for a preliminary identification, enabling the fruit owner or surveyor to assess the nature of the problem. Diagnosis can then be confirmed or modified by a specialist, making use of the microscope drawings and references together with appropriate practical techniques.

The majority of the photographs show samples of produce encountered during surveys of deteriorated consignments. A significant number show specimens received by the Plant Clinic of the Agricultural Development and Advisory Service (Ministry of Agriculture, Fisheries and Food), Cambridge, and I acknowledge with pleasure the help given by David Yarham, Michael Foley and, especially, Barry McKeown. Further plant material was kindly provided by Robert Beale, Paul Gans, Jeremy Sweet and Jane Thomas, of the National Institute of Agricultural Botany, Cambridge. Another valuable source was Dai Humphreys-Jones, who from time to time brought spectacular samples of mouldy vegetables. Excellent laboratory facilities were available in the Department of Applied Biology, under the professorship of Sir Joseph Hutchinson and later Sir James Beament. Technical advice on photography was provided by Gordon Revell, Don Manning and Bryan Barber; Allen Hilton lent his expertise in photomicrography. The remaining gaps in my slide collection were filled by borrowed photographs, for which I warmly thank the owners, acknowledged separately.

For permission to reproduce drawings from the invaluable *CMI Descriptions of Pathogenic Fungi and Bacteria* I am grateful to Professor D.L. Hawksworth, Director of the International Mycological Institute, Kew (formerly the Commonwealth Mycological Institute or CMI). Nomenclature is based on usage at the Institute, as defined in *Plant Pathologist's Pocketbook* (Ed. by A. Johnston & C. Booth, Commonwealth Agricultural Bureaux, 1983), in *Ainsworth & Bisby's Dictionary of the Fungi* (Ed. by D.L. Hawksworth, B.C. Sutton & G.C. Ainsworth, CAB International, 1983), in *Guide to Plant Pathogenic Bacteria* by J.F. Bradbury (CAB International, 1986) and in *A Dictionary of Plant Pathology* by P. Holliday (Cambridge University Press, 1989). A few synonyms are given in the text; some others may be traced via the index. For plant names and authorities, reference was made to E.E. Terrell's Checklist of Names for 3,000 Vascular Plants of Economic Importance (*USDA Agriculture Handbook* No. 505). Further information was obtained from *The Oxford Book of Food Plants* (Oxford University Press, 1969), *Evolution of Crop Plants* (Ed. by N.W. Simmonds, Longman, 1976), and J.W. Purseglove's *Tropical Crops. Dicotyledons* and *Tropical Crops. Monocotyledons* (Longman, 1968, 1972). Production data were taken from *FAO Production Yearbook* **41** (Food and Agriculture Organisation, 1988).

The text is elementary, technical terms being largely in parentheses. The aim is to provide the basic facts, with pointers to more detailed sources. No attempt has been made to recommend specific prophylactic chemicals, and local advice should be sought.

The references were chosen with the aim of including, for each main disease and disorder, a wide selection of journals, a range of countries and authors, one or two early classical papers (which are often rich in detail and illustration), together with recent reports describing current methods of control. The literature search was based on the CABI abstracting journals *Review of Plant Pathology* and (to a lesser extent) *Horticultural Abstracts*, which were scanned up till mid-1990. Holliday's *Fungus Diseases of Tropical Crops* (Cambridge University Press, 1980) was an invaluable source of key references up to and including 1978. *Post-Harvest Pathology of Fruits and Vegetables* (Ed. by C. Dennis, Academic Press, 1983) provided detailed reviews relating to temperate crops. Titles of papers in German, Dutch, French, Italian, Spanish and Portuguese have been retained in the original language; the use of English titles within square brackets denotes papers written in other foreign languages, usually with summaries in English. English summaries may in any case be consulted in the appropriate CABI abstracting journal; access is by means of the author-index or subject-index for the year in question or, more usually, the year following the reference date. For references from 1973 onwards, electronic access is also available (via the CABI database). The recent appearance of *Postharvest News and Information* (CAB Abstracts) is an indication of the increased emphasis now accorded to post-harvest studies.

Almost all of the original journals were available in the library of the Department of Applied Biology. (Following the closure of the Department in September 1989 the collection, renamed the Buttress Collection, was placed under the aegis of the Scientific Periodicals Library.) Other publications were consulted in the University Library, the Scientific Periodicals Library, and the libraries of the Botany School, the University Botanic Garden, the Shipowners' Refrigerated Cargo Research Association, Cambridge, the International Mycological Institute, Kew, and the Natural Resources Institute, Chatham. I thank all the librarians, and am especially indebted to Peter Filby for his continued interest and assistance.

I thank Awad Hassan Ahmed for help in compiling Appendix 3, which was first published in *The Storage and Transport of Fresh Fruit and Vegetables* (National Institute of Fresh Produce, 1981). The loan of facilities and equipment, by Anne Stow, Eric Miller and John Maunder, was much appreciated.

Many other generous individuals have helped me during the preparation of this book. It is not possible to mention them all, but they include Roy Burchill, Gill Butterfill, Tony Cooke, Desma Goddard, Glen Hartman, Lynn Hinton, Wilfred Hockfield, Greg Johnson, Stan Kays, Barbara Leonard, Dick Murfitt, Robert Noon, Felicity Proctor, Peter Sellar, Frederick Snowdon, Mary Snowdon, Henry Tribe, Janice Uchida, Stuart Wale, Elaine Watson and Gordon Wrigley.

I acknowledge with gratitude the part played by Dr Margaret Keay in offering detailed constructive criticism, in relation both to plant pathology and to grammatical construction. Sections of the completed text were checked by John Geeson, Geoff Hide, Francis Isenberg, Colin Leakey, Bill Ridgman and Peter Twiss, and the whole was finally read by John Turner. I am indebted to all these people for their helpful comments, but responsibility for errors remains mine alone, and I should be grateful if inaccuracies could be brought to my attention.

Lastly, I am indebted to Chris Hubbert and Anton Lawrencepulle for hauling me over the final hurdles, and to family and friends for their patience and support throughout.

Anna L. Snowdon
Wolfson College, Cambridge, 1991

Acknowledgements

Copyright relating to borrowed illustrations rests with the following individuals and/or institutions:

George S. Abawi, Cornell University **116**, **117**; Charles W. Averre, North Carolina State University **19**, **24**, **41**, **51**, **90**, **91**, **485**; Robert E. Beale, National Institute of Agricultural Botany, Cambridge **176**, **289**; Benny Bruton, United States Department of Agriculture, Lane, Oklahoma **16**, **17**, **33**; John Burden, Wisbech, Cambs (formerly of Tropical Products Institute, London) **464**, **468**; Nigel Cattlin, Holt Studios Ltd **246**; C.A. Clark, Louisiana Agricultural Experiment Station **479**, **483**, **490** (the last two previously published in *Compendium of Sweet Potato Diseases*, American Phytopathological Society 1988); W.S. Clark, Agricultural Development & Advisory Service, Cambridge **317**; Anthony W. Cooke, Queensland Department of Primary Industries **11**; Steven Dobson, Agricultural Development and Advisory Service, Cambridge **215**, **316**; Philip D. Dukes, United States Department of Agriculture, Charleston, South Carolina **496**, **497**, **498**, **503**, **504**, **506**; Peter G. Falloon, Department of Scientific and Industrial Research, New Zealand **214**, **217**; John D. Geeson, AFRC Institute of Food Research, Norwich **208**, **229**, **355**, **365**, **451**, **454**; Glen L. Hartman,

Asian Vegetable Research and Development Center, Taiwan **61, 62, 65, 67, 86, 115, 148, 149, 175, 209** (M.C. Chen); Geoffrey A. Hide/Robert L. Griffith, AFRC Institute of Arable Crops Research, Rothamsted Experimental Station **403**; Dai Humphreys-Jones, Ely, Cambs. **238, 429, 430**; Richard Kirby, formerly of Pennsylvania State University **87, 124** (previously published in APS Slide Set 33 No. 9 and Set 32 No. 12, American Phytopathological Society 1980); Derek H. Lapwood, Harpenden (formerly of AFRC Institute of Arable Crops Research, Rothamsted Experimental Station) **424**; S.M. McCarter, University of Georgia **52**; Barry M. McKeown, Processors' and Growers' Research Organisation, Peterborough **374**; Alan A. MacNab, Pennsylvania State University **15, 45, 66, 71, 83, 101, 165** (previously published in APS Slide Set 36 No.32, Set 36 No. 31, Set 33 No. 16, Set 33 No. 2, Set 37 No. 15, Set 33 No. 10, Set 32 No. 16, American Phytopathological Society 1980); W.J. Martin, Louisiana Agricultural Experiment Station **482** (previously published in *Compendium of Sweet Potato Diseases*, American Phytopathological Society 1988); Robert A. Noon, ICI Agrochemicals, Farnham (formerly of Tropical Products Institute, London) **456, 460, 463, 465, 469, 507, 511**; J.J. Ooka, University of Hawaii **467**; Roger C. Pearson, Cornell University **70**; D. Prusky, Volcani Center, Bet Dagan, Israel **400**; Z.K. Punja, Campbell Institute for Research and Technology, Davis, California **366, 367** (M. Ricker); Queensland Department of Primary Industries **121, 140, 167, 201, 499, 501, 505** (the four first-mentioned previously published in *A Handbook of Plant Diseases in Colour. Volume 1 Fruit and Vegetables*. 2nd ed. Queensland Department of Primary Industries 1982); L.H. Rolston, Louisiana Agricultural Experiment Station **489**; Arden F. Sherf, formerly of Cornell University **120, 178, 199, 227, 245, 325, 326, 337** (previously published in APS Slide Set 32 No. 18, Set 31 No. 30, Set 31 No. 15, Set 35 No. 30, Set 35 No. 21, Set 35 No. 4, Set 35 No. 3, Set 35 No. 8, American Phytopathological Society 1980); Ronald M. Sonoda, University of Florida **20**; Donald R. Sumner, University of Georgia **25, 42**; John D. Taylor, Horticulture Research International, Wellesbourne **135, 240, 303, 304, 305**; Jane Thomas, National Institute of Agricultural Botany, Cambridge **123, 159** (Michael Meadway), **164** (Andrew Tiley); UK Ministry of Agriculture, Fisheries and Food (Crown Copyright) **64, 81, 136, 225, 239, 258, 259, 261, 327, 345, 428**; United States Department of Agriculture **79, 97, 141, 154, 155, 163, 417** (previously published in *USDA Agriculture Handbooks*); Stuart J. Wale, Scottish Agricultural College, Aberdeen **151, 152, 281, 282, 283**.

Abbreviations

cf.	(confer) compare
cm	centimetre(s)
cv.	cultivar
f.sp.	forma specialis (sub-division of a species of fungus)
mm	millimetre(s)
pv.	pathovar (sub-division of a species of bacterium) (below the level of subspecies)
q.v.	(quod vide) which see
sp.	species (singular)
spp.	species (plural)
ssp.	subspecies
syn.	synonym
var.	variety
°C	°Celsius (Centigrade)
°F	°Fahrenheit

CMI Descr. *CMI Descriptions of Pathogenic Fungi and Bacteria*
Published by the International Mycological Institute in sets of 10, four sets per year. Each description gives morphological details of the pathogen (including spore measurements) together with information on hosts, diseases, geographical distribution, physiologic specialization, transmission, and relevant literature.

CMI Map *CMI Distribution Maps of Plant Diseases*
Published by the International Mycological Institute and updated as necessary. Each map shows the world distribution of the pathogen, together with references to the relevant literature.

Dedicated to the memory of

Frederick A. Buttress (1908-1984)

Librarian in the School of Agriculture (which became the Department of Applied Biology), University of Cambridge. Over a period of 45 years he assembled an incomparable collection of scientific journals from all parts of the world. It was the richness of this collection which provided the inspiration for the book.

INTRODUCTORY NOTE

A general introduction to the nature and causes of post-harvest deterioration is given in Volume 1, which emphasises that the post-harvest physiology of a commodity is directly related to the biological function of the plant part. Immature organs (such as flower buds and fleshy seeds) continue in a state of high metabolic activity after harvest, and their high respiration rates mean that they cannot be expected to remain fresh for more than a brief period, even under optimal conditions. At the other extreme, storage organs (such as mature roots and tubers), by virtue of their comparatively low respiration rates, may be kept in good condition for several months. Fruits can be classified according to their respiratory behaviour as 'climacteric' (characterised by a measurable rise in respiration rate at the onset of the ripening process) or 'non-climacteric' (showing a steady respiration rate at a given temperature). Superimposed on the intrinsic differences in storage potential between various commodities there are also differences in susceptibility to diseases and disorders. Diseases are caused by infectious micro-organisms, chiefly fungi and bacteria, while disorders are the result of abnormal or undesirable physiological changes such as those caused by nutritional deficiency, moisture loss or temperature stress. The general introduction in Volume 1 outlines the factors influencing post-harvest deterioration, including pre-harvest factors, harvesting and handling techniques, and conditions during storage and transport. The importance of the plant hormone ethylene is emphasised, both in relation to its role in the ripening of climacteric fruits and in relation to its capacity for inducing premature senescence in certain vegetables.

The term 'vegetable' encompasses a range of plant parts, and the common definition is a culinary one, denoting consumption as a savoury rather than as a dessert food. Some vegetables are (in the botanical sense) fruits, for example tomatoes, peppers and eggplants in the family Solanaceae, and cucumbers, marrows and squashes in the Cucurbitaceae. These commodities are sometimes referred to as 'fruit vegetables' or 'vegetable fruits'. Another example is avocado which, however, is more usually counted among the tropical fruits (see Volume 1). Conversely, rhubarb is loosely referred to as 'fruit' solely because it is customary to add sugar and eat it as a dessert; in reality, however, it consists of vegetative parts of the plant (succulent leaf stalks), and for that reason is often considered along with vegetables. Vegetables may also be shoots (for example, asparagus), leaves (lettuce), leaf stalks (celery), flower buds (globe artichokes), inflorescences (sprouting broccoli), seed-pods (peas and beans), bulbs (onions), roots (carrots), tubers (potatoes), rhizomes (ginger) or corms (taro). In the context of post-harvest physiology this is of fundamental significance.

---

1 BURTON W.G. (1982) *Post-harvest physiology of food crops.* London: Longman, 339 pp illus.

2 COOK A.A. (1978) *Diseases of tropical and subtropical vegetables and other food plants.* University of Florida: Institute of Food and Agricultural Sciences, 381 pp illus.

3 DENNIS C. (Ed.) (1983) *Post-harvest pathology of fruits and vegetables.* London: Academic Press, 264 pp.

4 DIXON G.R. (1981) *Vegetable crop diseases.* London: Macmillan, 404 pp illus.

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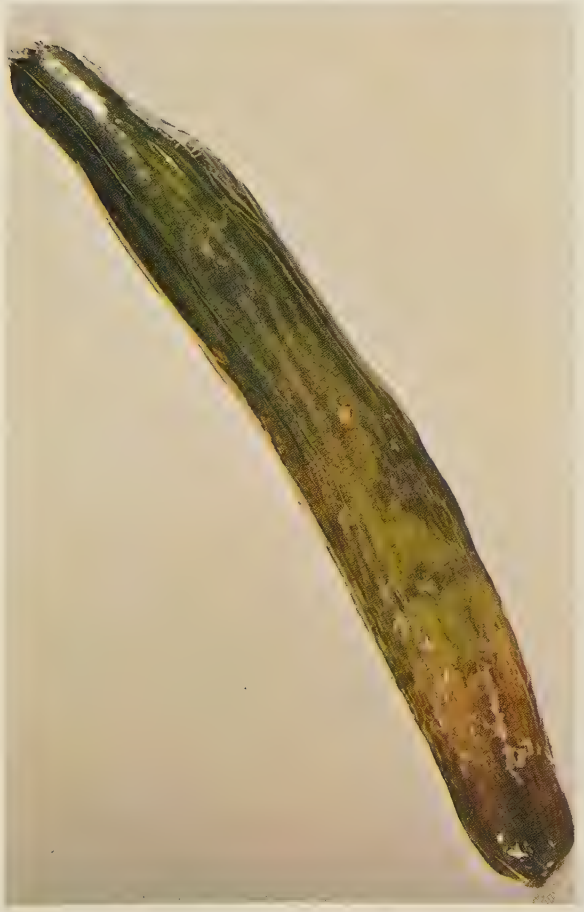
CHAPTER 1
CUCURBITS

The family Cucurbitaceae includes a number of important crop plants. Cucumbers, marrows, squashes, pumpkins and chayote fruits are popular vegetables in many countries. Melons and watermelons are generally eaten as dessert fruits but, because of their botanical affiliations, are usually considered along with cucumbers and other close relatives (10).

The **cucumber**, *Cucumis sativus* L., is thought to have originated in India. It has been cultivated since ancient times in Asia, Africa and Europe, and was well-known to the Romans. Columbus took seeds to the Americas in the 15th century. The Spanish word for cucumber is 'pepino'; confusingly, this same word is the common name for the 'exotic fruit' *Solanum muricatum* (see Volume 1).



1 Grey mould rot of cucumber (shrink-wrapped)

The cucumber thrives in the tropics and subtropics but has also become important in cooler latitudes where it is grown under glass or plastic. World production is over 12 million tonnes, the top ten producers being China, the USSR, Japan, Turkey, the USA, Romania, the Netherlands, Poland, Iraq and Egypt.

Cucumbers are usually eaten raw as a juicy salad vegetable. (There is also a substantial pickling industry using specially bred cultivars.) The fruits are harvested physiologically immature, at which stage they seem to exhibit a non-climacteric pattern of respiration (3). This means that their rate of respiration (which is fairly low) remains roughly constant at a given temperature. Furthermore, in healthy cucumbers held at moderate temperatures there is insignificant production of ethylene gas. Cucumbers are especially prone to moisture loss and also to decay (7). Common post-harvest diseases are cottony leak, black rot and bacterial soft rot (1,7). Control depends on appropriate cultural techniques, careful handling, and sometimes the use of chemicals (7). Prompt cooling is beneficial, especially if consignments are to be shipped long distances. 'Shrink-wrapping' in polyethylene film is very effective in minimising evaporation and weight-loss; waxing is an alternative method but carries a risk of suffocation (anaerobic respiration) and increased decay (7).

Storage life of cucumbers is intrinsically rather short (7). At temperatures above about 12° or 13°C (and especially in the presence of extraneous ethylene) they lose their dark green colour within a few days and begin to turn yellow (3). On the other hand, at lower temperatures there is a risk of chilling injury (q.v.). The optimal storage temperature is a compromise and is determined by the proposed duration of storage. At 10°C and high humidity, cucumbers can be kept for about 2 weeks. A lower temperature is feasible provided that the produce is utilised before chilling symptoms become apparent. Controlled atmosphere storage can give a useful extension of storage life for 'pickling cucumbers' awaiting processing.

The **melon**, *Cucumis melo* L., comprises a range of types, the common names of which have various connotations. The three varietal names are perhaps unjustified, since all the forms hybridise readily and there are many intermediate forms.

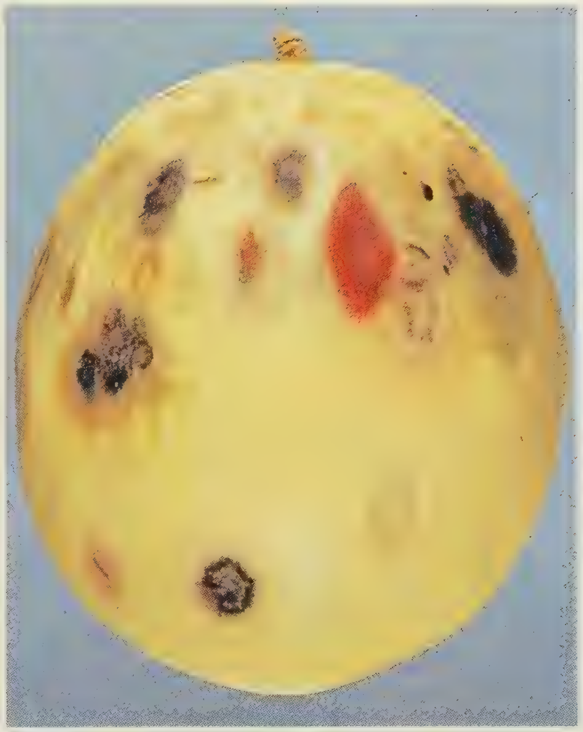
'Cantaloupes', *Cucumis melo* var. *cantalupensis*, are characterised by a thick rough rind with regularly spaced ribs but without surface 'netting'; Charentais is a typical cultivar, having a yellowish-green, slightly ribbed skin and fragrant orange flesh. True cantaloupes are popular in continental Europe; in the USA the term 'cantaloup' is applied to muskmelons (in either of the senses of that word) (q.v.).

'Muskmelons', *C. melo* var. *reticulatus*, are green or yellow with finely netted rinds and very shallow ribs; they have a musky fragrance. In American terminology 'muskmelon' encompasses all types of *C. melo* as distinct from watermelon (q.v.). In order to avoid ambiguity it might be helpful to use the term 'netted melons' for var. *reticulatus*.

'Winter melons', *C. melo* var. *inodorus*, mature late and can be stored for several weeks. They are oval or elongated, and do not possess a strong aroma. Important cultivars include Honey Dew with smooth ivory skin and green flesh, and Tendral which has a dark green, irregularly grooved rind and yellowish-green flesh.

The place of origin of the melon is not known with certainty but the evidence points to Africa. It subsequently spread into Asia, where many new forms developed (10). Melons were not cultivated in Europe until the late Middle Ages, except perhaps in Moorish Spain. Present world production is of the order of 9 million tonnes, one quarter of which is grown in China. Other important producers are Spain, the USA, Egypt, Iran, Romania, Japan, Italy, Mexico and Iraq.

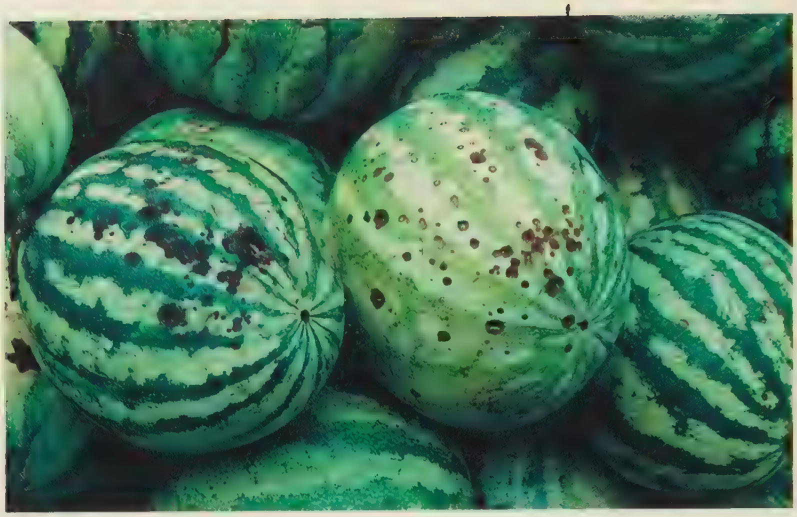
Melons are climacteric fruits but they have no starch reserves and cannot become significantly sweeter after harvest (10). Post-harvest ripening is restricted to softening of flesh and development of flavour volatiles (6). Picking is done by hand and if suitable illumination is available the work can be carried out at night rather than in the heat of the day; the benefits include enhanced retention of fruit quality and significant reduction in the cost of precooling for onward shipment. Cantaloupes and netted melons are best harvested at 'full-slip', when the fruit stalk is ready to separate naturally from the plant. At this stage they are approaching the climacteric peak, with the potential to develop full aroma (6). Harvesting at 'half-slip', when the stem-crack is still forming, allows for shipment to more distant markets yet still permits ripening to an acceptable quality. Winter melons behave in a different way and it is more difficult to judge their maturity. One indication is the changing colour of the 'ground-spot', the area of the fruit which rests on the ground. Honey Dew melons, even though harvested mature, do not necessarily have the capacity for self-ripening; in order to ensure uniform ripening in a batch of fruits it is necessary to apply ethylene treatment before shipment (6). Other post-harvest treatments may include a fungicide dip, some formulations being more effective if heated.



2 Various fungal lesions on Honey Dew melon

Prompt cooling of melons is highly desirable since it retards the breakdown of sugars (4). For netted melons the optimal temperature for storage and carriage is approximately 5°C, with a high humidity (or a plastic film wrap) to minimise moisture loss (4). Under such conditions they will keep for up to 2 weeks. Winter melons may need to be stored at 7° to 10°C. An exception is Honey Dew melons which have had the benefit of ethylene treatment; they show reduced susceptibility to chilling injury and can be held at 5°C. Some melon cultivars benefit from storage in controlled atmosphere, a 20% concentration of carbon dioxide being inhibitory to mould growth (9). This technique offers an alternative to the use of post-harvest fungicides. Common causes of deterioration in melons are fungal and bacterial blemishes and rots (2). For a detailed classical study of storage diseases of melons, see Reference 10 on page 49.

The **horned melon**, *Cucumis metuliferus* E. Mey. ex Naud., is usually classed as an 'exotic fruit', and began to receive attention only recently, first in New Zealand and subsequently in California. The fruits are oval, 10 to 15 cm long, and are characterised by numerous protuberances. On ripening, the skin turns yellow or orange but the jelly-like flesh remains green. 'Kiwano' is a patented name referring to a particular cultivar grown in New Zealand.



3 Anthracnose of watermelon

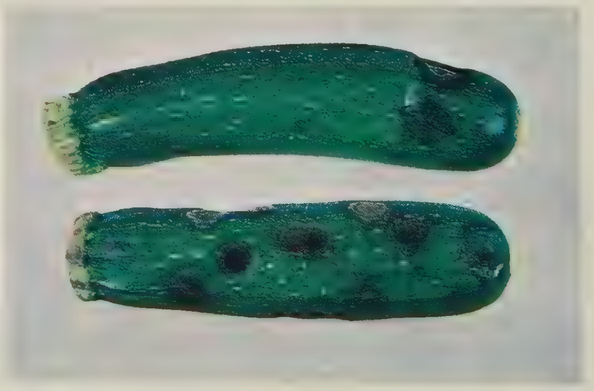
The **watermelon**, *Citrullus lanatus* (Thunb.) Matsum. & Nakai var. *lanatus*, is native to Africa. It has been cultivated in the Middle East for thousands of years. World production is currently nearly 30 million tonnes, grown mainly in China, Turkey, the USSR, Egypt, the USA, Iran, Japan, Italy, Spain and Syria. The watermelon is appreciated for its thirst-quenching properties and attractive appearance; some cultivars have yellow or orange flesh but most are deep pink with black seeds.

Until recently (Elkashif et al., 1989) it was believed that watermelons were climacteric fruits. Harvest maturity is judged on several criteria, including rind gloss and the colour of the ground-spot (10). The fruits should be cut from the plant with a sharp knife. If stem-end rot is likely to be a problem (1) it can be advantageous to disinfect the cut surface or to leave a long stalk so as to delay fungal advance into the fruit (10). Watermelons can be very large and heavy, up to about 20 kg, and even moderately sized fruits are difficult to handle without bruising (1). Small sized watermelons (between 2 and 5 kg) have recently been bred in the USA.

The optimal temperature for shipment and storage lies between 10° and 15°C, but shipment at ambient temperature is often feasible. Relative humidity is not critical, since watermelons do not readily lose moisture. They are sensitive to ethylene, however, and carriage with certain other commodities can lead to premature senescence (q.v.) of the watermelons. In the absence of ethylene they remain edible for as long as 2 or 3 months, but optimal quality is retained for 2 weeks at most.

Marrows, squashes and **pumpkins** are species of *Cucurbita*, a New World genus which has been cultivated in the Americas for several millennia, and was an important item in the diet of the Aztec, Inca and Mayan civilisations. After the Spanish Conquest seeds were carried to many parts of the globe (10). Current world production is about 6 million tonnes (this figure includes 'gourds'), grown mainly in China, Romania, Egypt, Argentina, Turkey, Italy, Japan, Syria, South Africa and Spain.

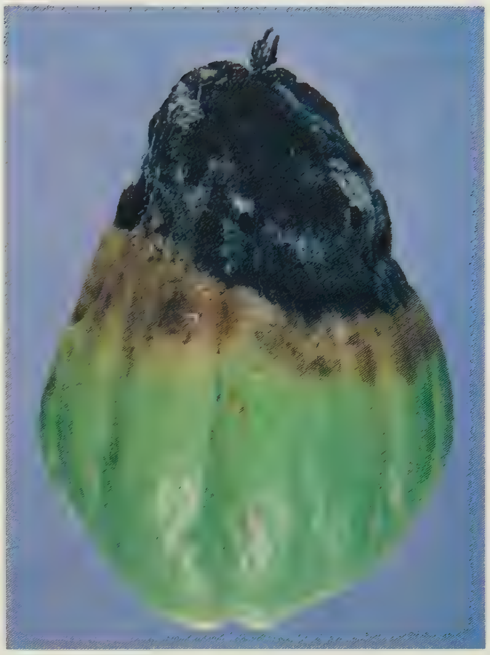
The terminology is confusing. 'Marrow' means different things in different countries. 'Squash' is an Amerindian word and is not understood as such in the UK (where the word, with a different derivation, means a fruit-flavoured drink). In North America summer squashes are the small immature fruits of *C. pepo* L., of which some cultivars are spherical or scalloped and others cylindrical, known as zucchini or courgettes in Europe. If the crop is harvested mature, the fully grown fruits are called winter squashes, an example of which is the large English 'vegetable marrow'. Other winter squashes are the mature fruits of *C. maxima* Duchesne var. *maxima*, *C. moschata* (Duschesne) Duchesne ex Poir. and *C. mixta* Pang. There is great variation, both within and between species, with respect to colour, shape and size of the fruits (10). Cultivars with coarse-grained flesh and a strong flavour are called pumpkins.



4 Cladosporium rot of summer squash

Storage conditions must take account of the fact that all are liable to chilling injury (q.v.). Cultivars differ in susceptibility. Recommendations for summer squashes vary between 7° and 10°C; at the higher temperature moisture loss is rapid (8). Chilling sensitivity can be reduced by controlled atmosphere storage, a carbon dioxide concentration of 5% permitting storage at 5°C (5). The tough-skinned mature fruits of winter squashes and pumpkins can be stored for weeks or months at moderate ambient temperatures, provided that atmospheric humidity is not excessive (10). Optimal conditions are a week or so above 20°C to permit wound-healing ('curing'), followed by storage at about 10° or 12°C and 75% relative humidity. In a study of several types of squash shipped to the New York market, bacterial soft rot was by far the commonest cause of loss (1).

The **chayote**, *Sechium edule* (Jacq.) Sw., is another New World species, and was well-known to the Aztecs (10). It is still important in Central America, and is also grown in other regions of the tropics and subtropics, including the West Indies, Australia, India and parts of Africa. Alternative names are choyote, chow-chow, cho-cho, choko and christophine, and the fruits can be boiled, stewed, fried or stuffed. They are pale green, up to 20 cm long, and shaped rather like a flattened pear with longitudinal furrows. Inside is a single large seed (which sometimes sprouts within the fruit). In order to avoid chilling injury (q.v.), chayotes should be shipped at 10° to 12°C, which confers a storage life of several weeks.



5 Black rot of chayote

The so-called **bitter cucumber** is not a true cucumber but another cucurbit, *Momordica charantia* L. The long green warty fruit is also called bitter gourd or balsam pear. It is especially common in India and the Far East. At tropical ambient temperatures it retains its quality for only a few days, while if stored or shipped at 12°C this period can be extended to 2 or 3 weeks.

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See also:

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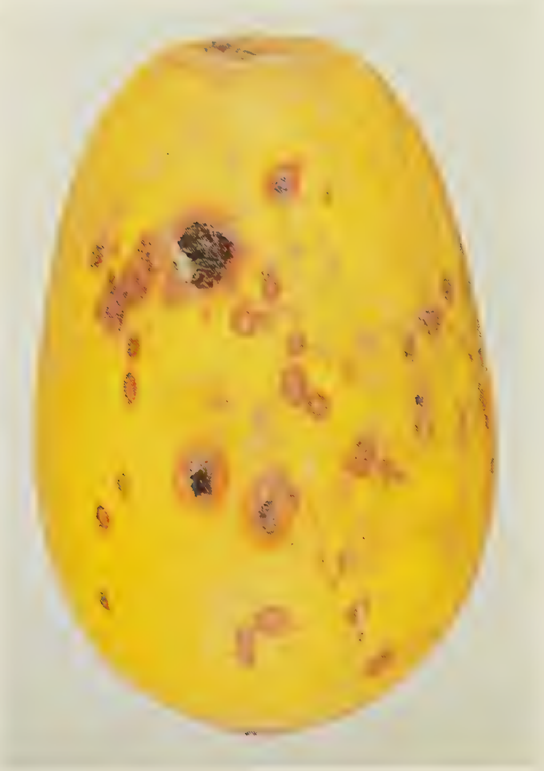
ALTERNARIA ROT of cucumbers, melons, watermelons, squashes and chayotes caused by

Alternaria alternata **(Fr.) Keissler**
Alternaria cucumerina **(Ell. & Ev.) Elliott**

6



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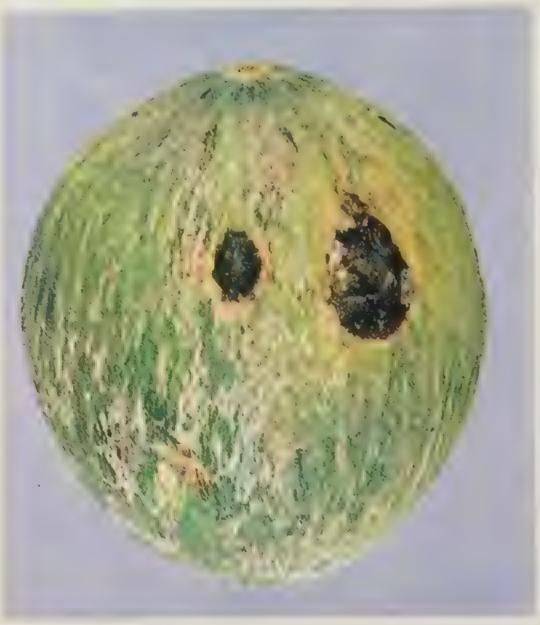
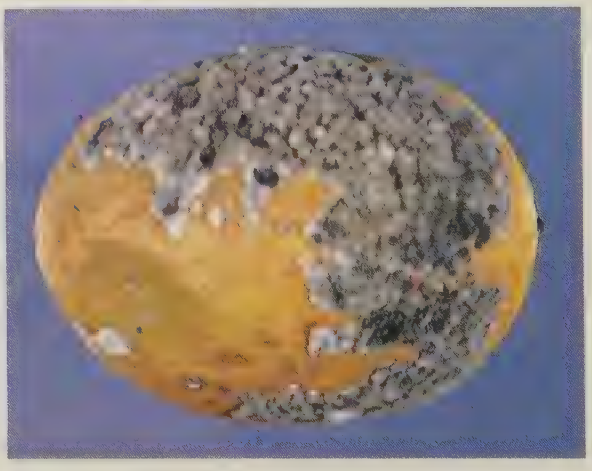


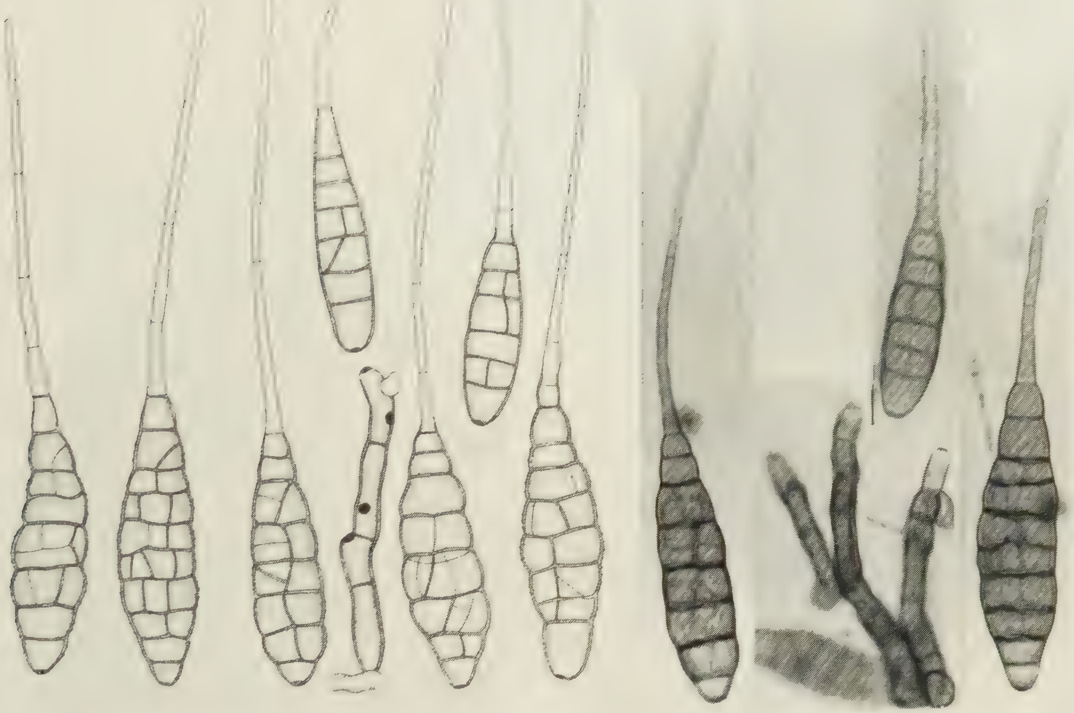
OCCURRENCE Alternaria rot probably occurs in all producing countries. *A. alternata* has been recorded on cucumbers and squashes in the USA (1,5), on melons and squashes in Israel (9), on melons in Australia (10), and on watermelons in India. *A. cucumerina* is usually more important as a cause of leaf-spot disease (3), but incidences of fruit infection have been reported on cucumbers and melons in the USA (3), melons in France (7), watermelons in Cyprus and squashes in India (2). Closely related fungi, *Stemphylium* spp. and *Ulocladium chartarum* (Preuss) Simmons, cause similar rots (4,6,8).

SYMPTOMS Lesions are circular to oval, and may become sunken. Affected skin at first appears bleached or brown but, under humid conditions, is rapidly covered by a dark mould and abundant olive-green to dark brown spores (5,7,8).

BIOLOGY The fungi probably survive in crop debris (3), and spores can be disseminated by air currents. Leaf blight caused by *A. cucumerina* can result in defoliation and exposure of fruits to the sun. Fruits weakened by sunscald are predisposed to attack by either species of *Alternaria* (3). Similarly, if fruits are stored for too long, or at too low a temperature with consequent chilling injury (q.v.), they become especially prone to alternaria decay, and suffer multiple lesions, probably centred on natural openings (stomata) in the skin (5). The author has observed such symptoms on chayotes which had been shipped from Caribbean countries at too low a carriage temperature. Species of *Alternaria, Stemphylium* and *Ulocladium* can also infect fruits through the cut stem or via injuries in the rind (6,10).

8





9 *Alternaria cucumerina* (from CMI Descr. No. 244, Ellis & Holliday 1970) Conidia and conidiophores × 500

CONTROL It may be advisable to treat the seed with a fungicide before planting (3). Control of *A. cucumerina* must be carried out during the growing season, by the use of sprays (2,7). Post-harvest decay by all these fungi can be minimised by careful handling and packing so as to prevent unnecessary injury (6), by the use of an appropriate fungicide dip (10), and by storage at an appropriate temperature (5). Fruits should not be kept for too long, especially if they have not been harvested at optimal maturity (3).

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ANTHRACNOSE of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes caused by

Glomerella cingulata **(Stonem.) Spauld. & v. Schrenk**
Conidial state: *Colletotrichum gloeosporioides* f. sp. *cucurbitae*
Glomerella magna **S.F. Jenkins & N.N. Winstead**
Conidial state: *Colletotrichum* sp.

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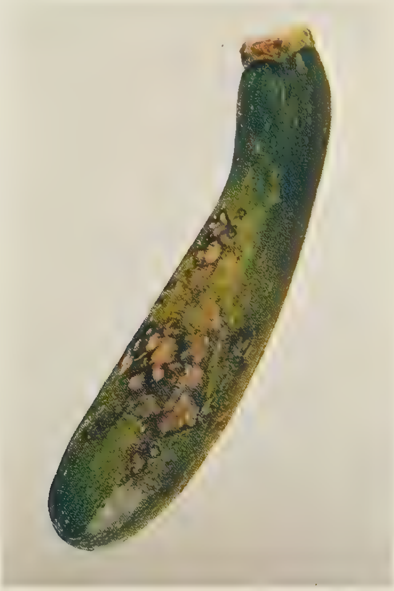

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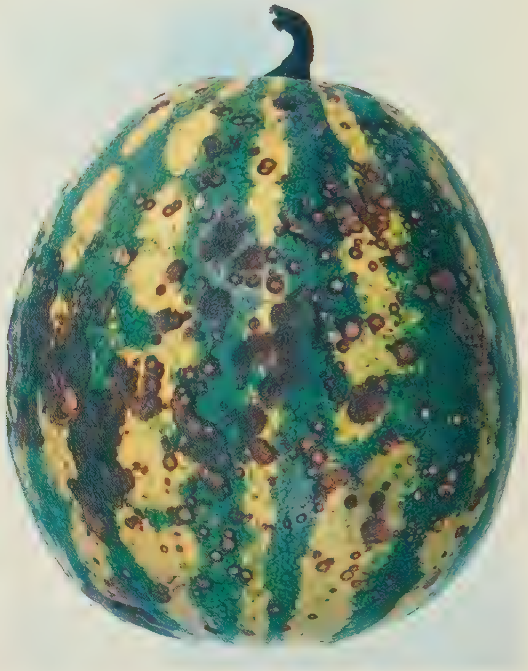
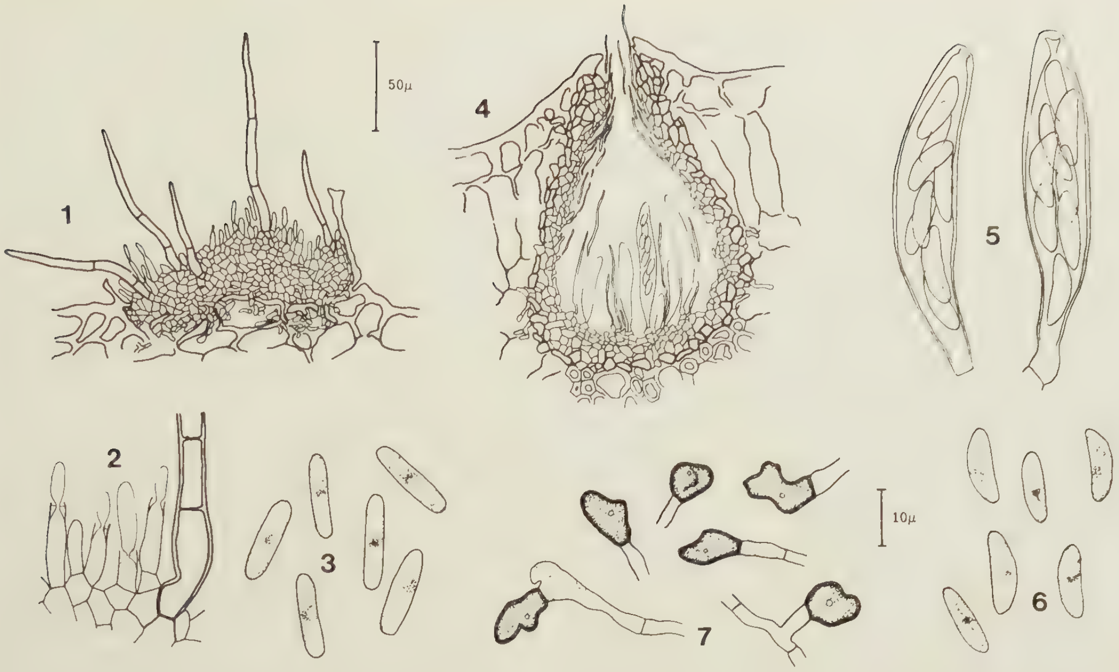


OCCURRENCE Anthracnose is usually caused by *Colletotrichum gloeosporioides* f. sp. *cucurbitae* (often known as *C. lagenarium* (Pass.) Ell. & Halst. or *C. orbiculare* (Berk. & Mont.) v. Arx) which can be especially virulent on cucumbers, melons, watermelons and chayotes (2,7,8). Serious losses have been reported from many countries, including the USA (3,10), Brazil (8), the Netherlands (2), India (1), Brunei (9) and Japan (6). Marrows, squashes and pumpkins are generally resistant to attack by this fungus. A related species of *Colletotrichum* (sexual state *Glomerella magna*) has been recorded in the USA on melons, watermelons, squashes and pumpkins but is of little importance on cucumbers (5).

SYMPTOMS Conspicuous dark circular spots, often very numerous, develop on the fruit surface. The lesions become sunken and sometimes cracked and, under humid conditions, characteristic masses of pinkish spores (conidia) are formed (2,3,7). Disfigurement can be severe; the common anthracnose fungus is usually confined to the skin of the fruit, though the flesh may subsequently be invaded by secondary bacteria causing a soft rot (q.v.). Lesions caused by *G. magna* may develop concentric rings, and the fungus is capable of colonising the entire fruit (5).

BIOLOGY The anthracnose fungi survive in dead plant material in field or glasshouse, when they may produce the sexual stage (perithecia giving rise to ascospores) (2,5). It is the asexual stage (acervuli giving rise to conidia) which is important in infection of the growing crop, and spores may be transmitted by insects (4) as well as by rain or irrigation water (3). Infection is particularly severe after prolonged wet periods (1), since moisture permits spore germination and direct penetration of undamaged leaves, stems and fruits (6). Disease development is rapid at temperatures between 20° and 30°C (1,7,10).

CONTROL Strict hygiene is necessary in field or greenhouse. All crop debris should be removed and destroyed, and in greenhouses any rotten woodwork should be chemically treated in case it is harbouring the fungus. It may be advisable to treat the seed before planting, as there is evidence of seed transmission (4). It has been demonstrated that disease severity can be substantially reduced by inoculating young plants with the fungus so that they acquire resistance during the early stages of growth. Fungicide sprays have proved useful against



12 *Glomerella cingulata* (from CMI Descr. No. 315, Mordue 1971) 1 acervulus 2 conidiophores 3 conidia 4 perithecium 5 asci 6 ascospores 7 appressoria

anthracnose, and disease forecasting helps to ensure that they are well-timed (1,10). Alterations to irrigation regimes can help minimise the likelihood of infection; frequent overhead sprinkling, for example, can result in epidemics (1,3,6,9). Plant breeders have investigated means of developing new cultivars (especially of cucumber and watermelon) which are resistant to the several races of anthracnose fungi which exist (10). Resistant pickling cucumbers have been bred in the USA.

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BACTERIAL ROTS of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes

Several species of bacteria may be present in the internal tissues of healthy fruits (3). Under certain circumstances, however, bacteria can multiply and cause serious disease.

Bacillus polymyxa **(Prazmowski) Mace** was found on Israeli melons, causing large brown spots which were sometimes hard and sometimes soft (9).

Erwinia carotovora **ssp.** ***carotovora*** (page 302) has caused soft rotting of melons grown in greenhouses in Japan, and also a watery rot of stored melons in Brazil. In the USA the same organism (under different names) was recorded on melons, squashes and pumpkins, and found to be capable of causing soft rotting in wounded cucumbers and watermelons (1,2). Insect damage and mechanical injuries predispose fruits to infection. An uneven water supply can result in cracking of fruit and bacterial invasion (2). The flesh disintegrates into a soft mass and the skin ruptures further, releasing infective juices and a foul odour.

Erwinia ananas **Serrano** has been found on melons imported into the USA from Ecuador, Guatemala and Venezuela, as well as on melons grown in California and Texas (10). It is a yellow-pigmented bacterium and causes smooth, firm, brownish spots. These lesions are slow to develop and may remain superficial rind blemishes, but they can be up to 4 cm across and constitute a grave disfigurement. Furthermore, some strains of the bacterium are sufficiently virulent as to cause an invasive soft rot extending into the seed cavity.

Erwinia **spp.** have often been noted on melons in the southern USA, causing dark, circular, water-soaked lesions on the skin, and numerous hard dry brown areas within the rind and also scattered in the flesh (8). In watermelons there are usually no external symptoms but, when the fruit is cut, hard brown areas are apparent within the rind. **E. herbicola** (page 240) is one of a range of bacteria which may affect watermelons in this way (3).

Pseudomonas burgeri (the name is not valid) has been reported on greenhouse cucumbers in Romania (4). Under warm humid conditions circular sunken spots form on the fruit, and the bacteria invade the vascular system, causing a soft rot of the entire fruit. Subsequent studies suggest that the organism is a virulent strain of ***Pseudomonas syringae*** **pv.** ***lachrymans***, the cause of angular leafspot and bacterial spot (q.v.).

Xanthomonas campestris **pv.** ***cucurbitae*** **(Bryan) Dye** has caused scab-like spots on melons in Japan (7) and a severe blemish of pumpkins in Australia (5). The disease is seed-transmitted and can be controlled by treating the seed with a bactericide.

Xanthomonas campestris **pv.** ***melonis*** **Neto, Sugimori & Oliveira** has been found in melons in Brazil (6). Small dark depressed spots coalesce to form large areas of soft watery tissue which is usually free from unpleasant odour.

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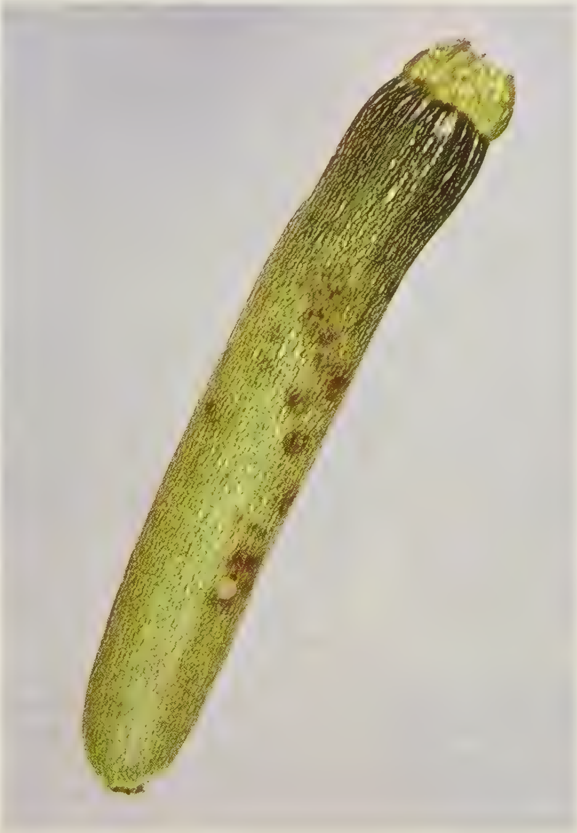
BACTERIAL SPOT of cucumbers, melons, marrows and squashes caused by

Pseudomonas syringae* pv. *lachrymans **(Smith & Bryan) Young, Dye & Wilkie**

OCCURRENCE Bacterial spot is important in warm regions such as the central and southern USA (4,5), Israel (1) and Japan. In Italy it has caused severe losses in marrows grown under glass (2).

SYMPTOMS Typical symptoms on the fruit surface are circular, sunken, watersoaked spots which may be very numerous. The interior of the fruit may undergo a rapid soft rot, with browning of the vascular tissue (3,4,5).

BIOLOGY The organism survives on debris from an infected crop, and can also persist in the soil in the presence of crops other than cucurbits (1). The bacteria are disseminated by rainsplash or irrigation water, and cause 'angular leafspot' of the growing plant (2). Infection occurs via the natural openings (stomata) in leaves and fruits, and a bacterial exudate may ooze from the lesions (5). Under warm, moist conditions the disease spreads rapidly, the optimal temperature for bacterial multiplication being approximately 25°C. In hotter drier conditions disease development is arrested and the lesions dry out, taking on a chalky appearance. If the fruits are handled gently at harvest and marketed without delay, disease symptoms are confined to superficial disfigurement. It frequently happens, however, that fruits appear in good condition or only slightly blemished at the time of packing, but vibration suffered during the transit period precipitates rapid development of bacterial soft rot (5). Juices leak out, contaminating and infecting adjacent healthy fruits. It has also been found that this bacterium may first enter the fruit at the stem-end, as a result of injury during the growing season or via the cut stalk at harvest time (3). Soft spots on the fruit surface then result from lateral movement of bacteria outward from the invaded vascular system. Seeds are infected, thereby transmitting the disease to the next crop (5).



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CONTROL Crop debris should be removed and destroyed, and seed treated with a bactericide before planting. Chemicals may also be applied to the growing crop (2). The likelihood of infection can be minimised by employing measures which reduce humidity within the crop, for example the use of trickle irrigation as opposed to sprinklers, or the use of hot air dehumidifiers in greenhouses. If harvested fruits show signs of infection they should be marketed locally without delay.

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1 KRITZMAN G. & ZUTRA D. (1983) Survival of *Pseudomonas syringae* pv. *lachrymans* in soil, plant debris, and the rhizosphere of non-host plants. *Phytoparasitica* **11**, 99–108.

2 MARRAS F. & CORDA P. (1973) *Pseudomonas lachrymans* (Smith et Bryan) Carsner, agente della "maculatura angolare" delle foglie e del "marciume" dei frutti dello zucchino (*Cucurbita pepo* L.). *Studi Sassaresi* III **21**, 817–827.

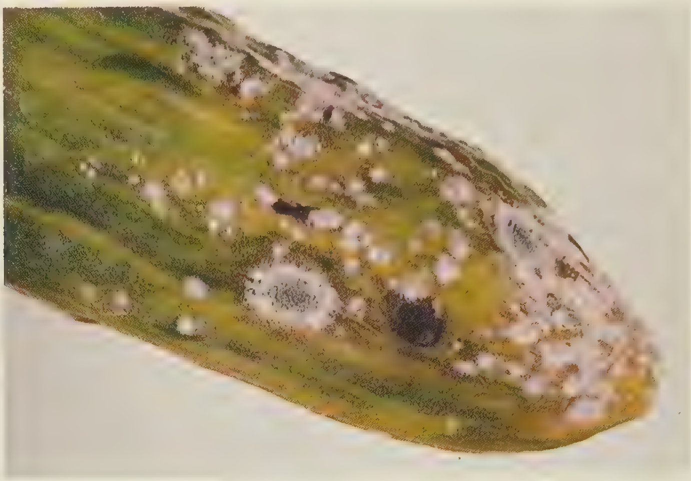
3 POHRONEZNY K., LARSEN P.O. & LEBEN C. (1978) Observations on cucumber fruit invasion by *Pseudomonas lachrymans*. *Plant Disease Reporter* **62**, 306–309.

4 SMITH M.A. (1946) Bacterial spot of honeydew melon. *Phytopathology* **36**, 943–949 illus.

5 WEBER G.F. (1929) Angular leaf spot and fruit rot of cucumbers caused by *Bacterium lachrymans* E.F.S. & Bry. *Bulletin of Florida Agricultural Experiment Station* No. 207, 32 pp illus.

BLUE MOULD ROT of cucumbers and melons caused by ***Penicillium*** **spp.**

14



OCCURRENCE Blue mould rot is a common disease of cucurbits, although published records appear to be confined to melons and cucumbers. *P. italicum* has been found on Bulgarian cucumbers (2) and *P. oxalicum* on Canadian cucumbers (4). Blue mould rot has been observed on melons from Israel (1), South Africa (3), the USA, Mexico (5), Ecuador and Peru. Species identified include *P. crustosum* Thom, *P. cyclopium* Westling, *P. pallidum* Smith and *P. viridicatum* Westling. The author has recorded *P. expansum* (page 88) on Spanish melons imported into the UK.

SYMPTOMS On melons lesions are at first circular to oval, often watersoaked and with a tendency to rupture; blue-green spores are produced in the cracks so formed, and there is a typical musty odour (5). Some strains of these fungi form restricted lesions, while others spread over much of the fruit and cause a wet collapse (3,5). In fresh cucumbers there may be multiple small circular lesions covered with spores while, in pickled cucumbers, heat-resistant enzymes produced by some species of *Penicillium* are responsible for undesirable softening of the flesh (4).

BIOLOGY Many penicillia are present in soil and in the atmosphere, and fruit is probably contaminated at the time of harvest or in the packhouse. Infection is typically via wounds or directly through skin which has been weakened by chilling injury or prolonged storage.

CONTROL Strict hygiene should be practised in field, greenhouse and packhouse. Culled fruits should be buried before blue mould can proliferate on them, and boxes used for picking should be regularly disinfected. Fruits should be handled with care so that injuries are minimised. Harvested fruits can be subjected to irradiation (1) or dipped in a fungicide (2), and should not be kept in store for too long (5).

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1 BARKAI-GOLAN R., KAHAN R.S. & TEMKIN-GORODEISKI N. (1968) Sensitivity of stored melon fruit fungi to gamma irradiation. *International Journal of Applied Radiation and Isotopes* **19**, 579–583.

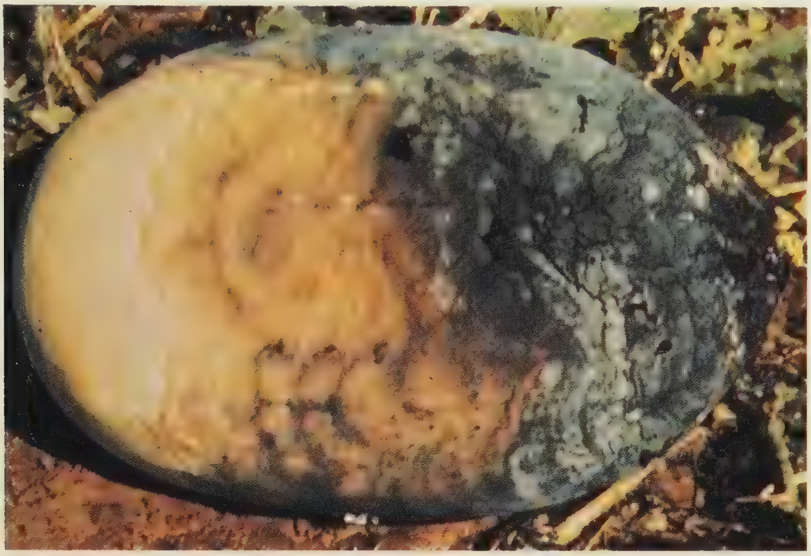
2 GEORGIEVA M. & KOTEV S. (1977) [Rot of cucumber fruits during transportation and its control.] *Bulgarski Plodove, Zelenchutsi i Konservi* No. 6, 23–26.

3 RATTRAY J.M. (1939) In *Report of the Low Temperature Research Laboratory, Cape Town, 1937–1938*, pp 55–65.

4 RAYMOND F.L., ETCHELLS J.L., BELL T.A. & MASLEY P.M. (1959) Filamentous fungi from blossoms, ovaries, and fruit of pickling cucumbers. *Mycologia* **51**, 492–511.

5 WIANT J.S. (1937) Investigations of the market diseases of cantaloups and Honey Dew and Honey Ball melons. *Technical Bulletin of the United States Department of Agriculture* No. 573, 47 pp illus.

BOTRYODIPLODIA ROT of cucumbers, melons, watermelons, squashes and pumpkins caused by ***Botryodiplodia theobromae*** Pat.



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OCCURRENCE This disease, formerly known as diplodia rot, can cause serious losses in warm regions such as the southern USA (1,4), Chile (5), India (2) and Egypt (3).

SYMPTOMS Lesions are initially buff-coloured, and the affected area is spongy with a water-soaked margin (5). Dark grey mould strands may develop on the surface. The fungus also produces a tough layer beneath the skin of the fruit, causing it to become severely wrinkled. Minute black bodies (pycnidia) are usually produced, and a sour odour accompanies the decay. Lesions may occur anywhere on the fruit, but are frequently found at the stem-end (1,4). Invasion of the seed-cavity results in blackening of the seed (2). Cucumbers, melons and watermelons tend to undergo a dry rot, but pumpkins suffering from this disease become soft and lose their shape (3).

BIOLOGY The fungus, which is common in soil and on crop debris, exists in the asexual state (pycnidia giving rise to conidia) and the spores are dispersed by wind and water (page 371). The growing crop may suffer from stem blight (1,3), with symptoms indistinguishable from those caused by *Didymella bryoniae* (q.v.). Fruits can only be infected via a wound (5), including that made by cutting the stem at harvest (1). The optimal growth temperature for the fungus is approximately 30°C and it grows scarcely at all below 10°C (5).

CONTROL It may be necessary to use a fungicide spray on the growing crop (1). Irrigation should be carefully controlled (3). Care should be taken to minimise injury during harvesting and handling, and a post-harvest fungicide should be applied, either as a paste on the cut stem (5) or as a dip to protect the whole fruit. This fungus can be effectively held in check by refrigeration.

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1 BERAHA L., TOWNER D.B. & CAMP T.H. (1976) Stem gumming and blight and stem end rot of Texas cantaloups caused by *Diplodia natalensis*. *Plant Disease Reporter* **60**, 420–422 illus.

2 MAHOLAY M.N. & SOHI H.S. (1982) *Botryodiplodia* seed rot of bottlegourd and squash. *Indian Journal of Mycology and Plant Pathology* **12**, 32–36.

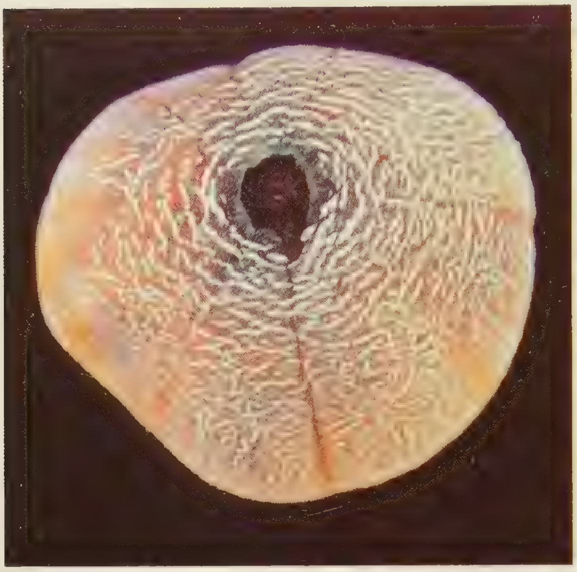
3 SATOUR M. & SHINNAWY S. (1977) *Botryodiplodia* fruit rot of pumpkin in Egypt. *Agricultural Research Review* **55**(2), 113–115.

4 TAUBENHAUS J.J. & EZEKIEL W.N. (1934) Stem-end rot of watermelons. *Annual Report (1933) of Texas Agricultural Experiment Station* No. 46, 88.

5 WIANT J.S. (1937) Investigations of the market diseases of cantaloups and Honey Dew and Honey Ball melons. *Technical Bulletin of the United States Department of Agriculture* No. 573, 47 pp illus.

CHARCOAL ROT of melons, watermelons, marrows, squashes and pumpkins caused by ***Macrophomina phaseolina*** **(Tassi) Goid.**

16



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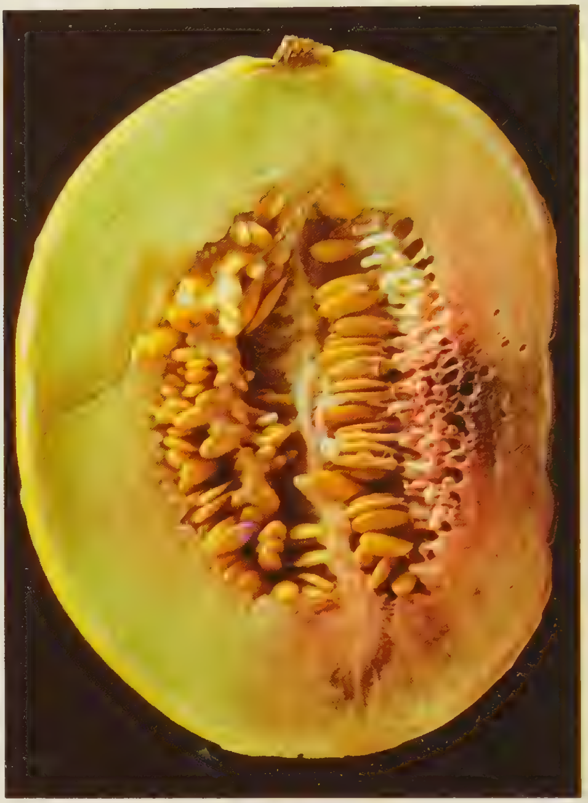
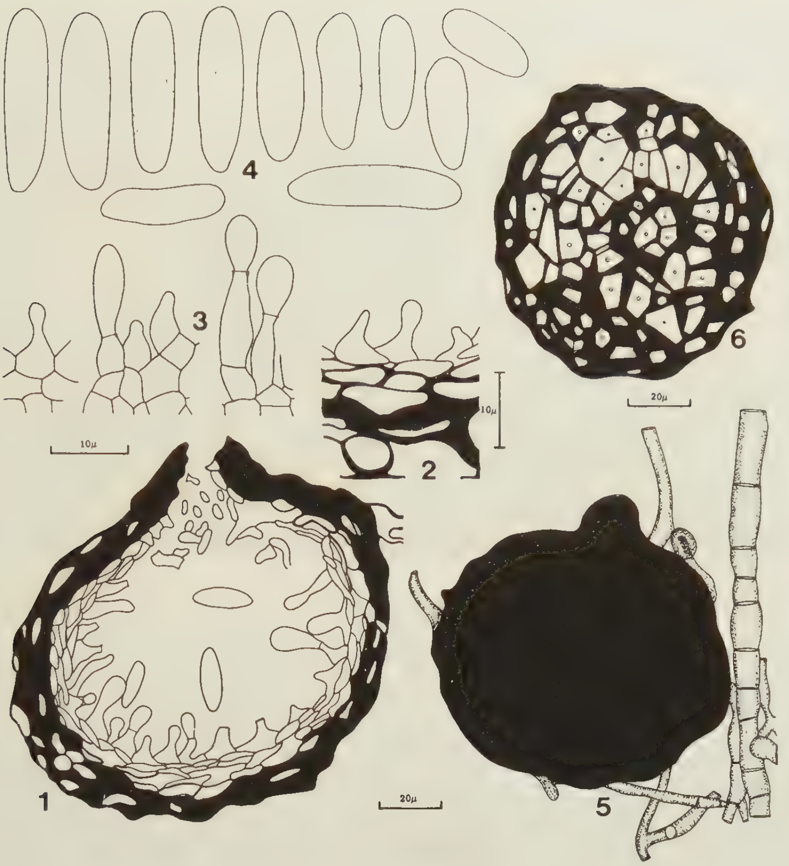


OCCURRENCE The fungus is widespread in the tropics and subtropics, and can cause epidemics during hot dry seasons (10). Charcoal rot is of particular importance on melons, and has been found on rotted fruits in India (9), Israel (3,7), the southern USA and Chile (2). It has also been recorded on watermelons and marrows in India (9) and on watermelons, squashes and pumpkins in the southern USA (10).

SYMPTOMS A watersoaked spot appears on the skin and, under favourable conditions, enlarges rapidly, the affected area being firm in texture with an indefinite boundary. A bright purple or pink coloration may be associated with the lesion, both externally and internally (2) (cf. fusarium rot, q.v.). As the decay advances, the skin becomes wrinkled and there is profuse development of very minute spherical black bodies (microsclerotia). They are so small as to appear almost powdery, hence the name 'charcoal rot'. This superficial crust tends to rupture, exposing watery flesh which emits a pungent odour and eventually turns black. Sometimes infected fruits become hollow shells, of which the interior is also blackened (10).

BIOLOGY Under dry conditions the small resting bodies (microsclerotia) persist in the soil (1,5,8) or in the roots of plants (4). Infection of growing crops is most severe during hot weather (7), the optimal growth temperature for the fungus being 30° to 35°C. Roots are invaded (3) and the fungus then progresses into the stems (2). Fruits may become infected before harvest, either via the stem-end or from the soil through a wound in the skin of fruit in contact with the ground (9). Infection of fruits may also occur at the time of harvest. Occasionally the fungus produces the asexual state (pycnidia giving rise to conidia) on stems (2) or on fruits (9). Seeds are often infected, thereby transmitting disease to the next crop (3,6,7).

CONTROL Soil can be disinfected by drenching with a fungicide; however, it is not easy to kill this fungus (5). Clean or treated seed should be planted (7). Control has been achieved by coating seeds with a preparation containing an organism which is antagonistic to the fungus but otherwise harmless (biological control) (3). If rain is infrequent, irrigation should be carried out regularly (10), firstly so that the plants are not weakened by drought stress (3) and secondly because the charcoal rot fungus survives poorly in wet soil (1,8). Fruits can be protected by means of a post-harvest fungicide dip and by the use of refrigerated storage.



18 *Macrophomina phaseolina* (from CMI Descr. No. 275, Holliday & Punithalingam 1970) 1 pycnidium 2 part of pycnidial wall and conidiophores 3 conidiophores and young conidia 4 conidia 5 sclerotium 6 section of sclerotium

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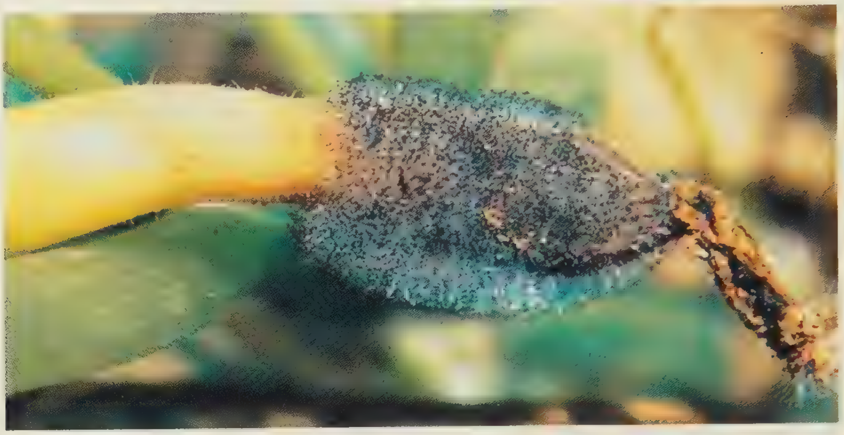
1 BANERJEE S., MUKHERJEE B. & SEN C. (1983) Survival of mycelia and sclerotia of *Macrophomina phaseolina* in soil: influence of moisture and temperature. *Indian Journal of Plant Pathology* **1**, 20–23.
2 CARTER W.W. (1979) Importance of *Macrophomina phaseolina* in vine decline and fruit rot of cantaloup in south Texas. *Plant Disease Reporter* **63**, 927–930 illus.
3 ELAD Y., ZVIELI Y. & CHET I. (1986) Biological control of *Macrophomina phaseolina* (Tassi) Goid. by *Trichoderma harzianum*. *Crop Protection* **5**, 288–292.
4 GHAFFAR A. & AKHTAR P. (1968) Survival of *Macrophomina phaseolina* (Maubl.) Ashby in cucurbit roots. *Mycopathologia et Mycologia Applicata* **35**, 245–248.
5 HARTZ T.K., CARTER W.W. & BRUTON B.D. (1987) Failure of fumigation and solarization to control *Macrophomina phaseolina* and subsequent muskmelon vine decline. *Crop Protection* **6**, 261–264.
6 MAHOLAY M.N. & SOHI H.S. (1983) *Macrophomina* seed rot of bottlegourd, squash and muskmelon. *Indian Journal of Mycology and Plant Pathology* **13**, 192–197.
7 REUVENI R., NACHMIAS A. & KRIKUN J. (1983) The role of seedborne inoculum on the development of *Macrophomina phaseolina* on melon. *Plant Disease* **67**, 280–281.
8 SHOKES F.M., LYDA S.D. & JORDAN W.R. (1977) Effect of water potential on the growth and survival of *Macrophomina phaseolina*. *Phytopathology* **67**, 239–241.
9 SINGH R.S. & COLHOUN J.S. (1972) Studies on charcoal rot of cucurbits. *Plant Disease Reporter* **56**, 115–118 illus.
10 YOUNG P.A. (1949) Charcoal rot of plants in east Texas. *Bulletin of Texas Agricultural Experiment Station* No. 712, 33 pp illus.

CHOANEPHORA ROT of cucumbers, watermelons, marrows, squashes and pumpkins caused by

Choanephora cucurbitarum **(Berk. & Rav.) Thaxt.**

19



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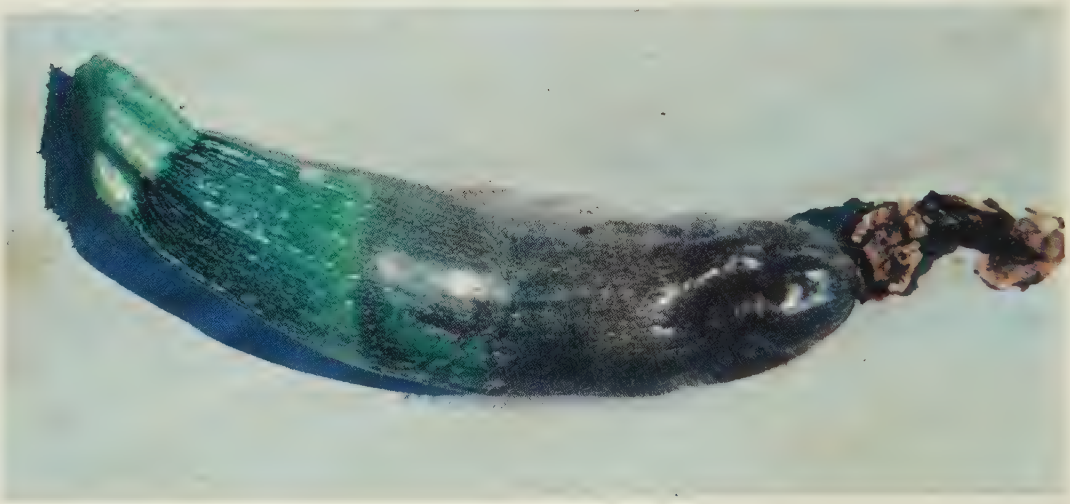
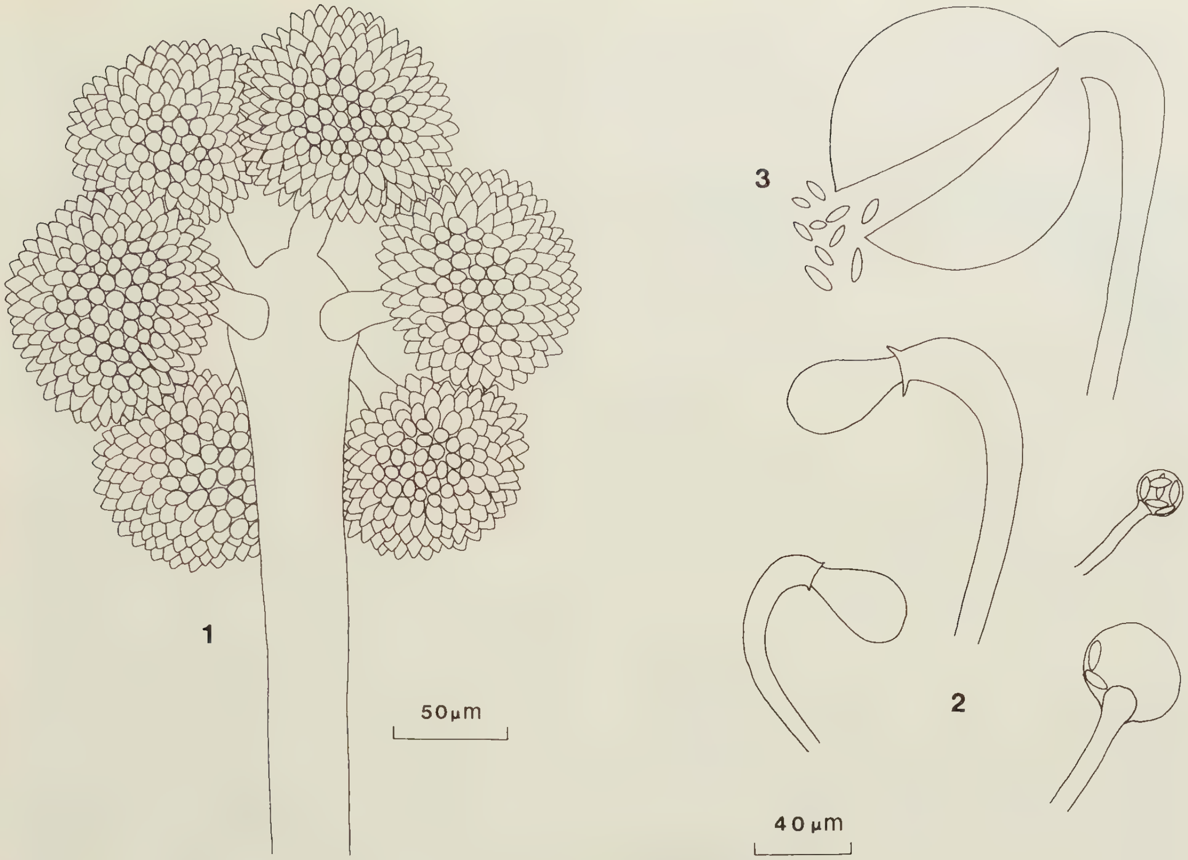


OCCURRENCE This fungus has caused severe losses of marrows in Egypt (1,3) and Romania (8), squashes in the USA (10) and pumpkins in the USA (2) and India (7). It is less important on cucumbers (9) and watermelons (6). A closely related fungus (*Cunninghamella echinulata* (Thaxt.) Thaxt. ex Blakeslee) has been found on pumpkins in India (4).

SYMPTOMS A soft wet rot develops, usually starting at the blossom-end of the fruit (1,8). Under humid conditions there is a luxuriant growth of whitish mould, which takes on a characteristic metallic lustre (10). Numerous spore-heads develop (giving rise to sporangiospores) and turn from white to brown to purplish-black. Decay is very rapid at temperatures between 25° and 30°C. In a cooler, drier environment there may be no development of mould on the surface, and the fruits merely turn brown (8).

BIOLOGY As well as producing two types of asexual spores (sporangiospores and conidia) the fungus can also exist in the sexual state (zygospores) or as resting spores (chlamydospores) in the soil (2,6,8,10). Sporangiospores are disseminated by wind and insects and, under warm wet growing conditions, dying flowers become infected (10). From the female flowers the fungus subsequently invades the fruit, and decay may occur in the field or during marketing (1,8).

CONTROL Crop rotation is beneficial, provided that the cucurbit crop does not follow other susceptible crops such as legumes, peppers, cauliflowers, okra or cotton (8). Since disease development is favoured by excessive moisture, care should be taken to ensure good drainage, moderate irrigation, a low crop density and freedom from bushy weeds (8). If wet conditions are unavoidable, a fungicide spray can



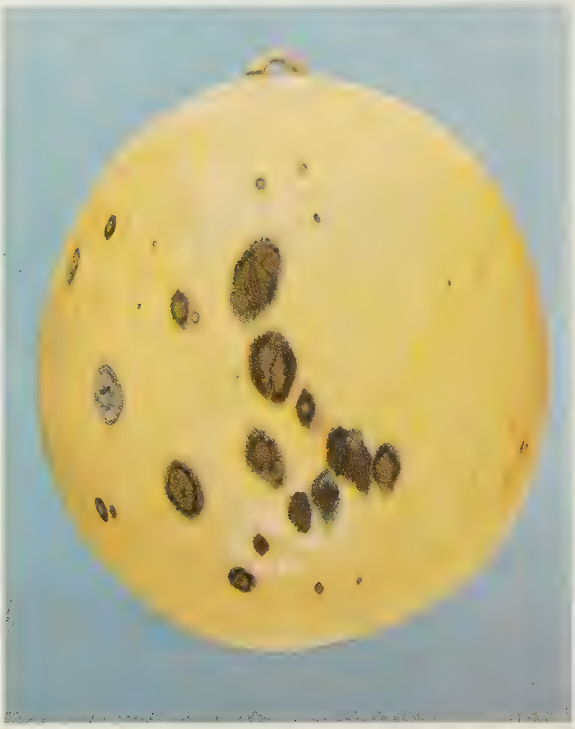
21 *Choanephora cucurbitarum* (from *Mycological Papers* No. 152, Kirk 1984) 1 mature sporangioliferous sporangiophore bearing six fertile vesicles 2 sporangial fructifications 3 sporangiospores

be used (3). If infection is noticed in the growing crop, all affected fruits should be removed and destroyed, and the plants burned after harvest (8). The use of refrigerated storage serves to minimise decay in fruits harvested in apparently sound condition.

1 ABD-EL-REHIM M.A., ELAROSI H. & MICHAIL S.H. (1964) Choanephora fruit rot of vegetable marrow in the U.A.R. (Egypt). *Alexandria Journal of Agricultural Research* **12**(2), 123–128 illus.
2 BARNETT H.L. & LILLY V.G. (1956) Factors affecting the production of zygospores by *Choanephora cucurbitarum*. *Mycologia* **48**, 617–627.
3 EL-HELALY A.F., ASSAWAH M.W., ELAROSI H.M. & WASFY E.-E.H. (1968) Studies on the control of *Choanephora* blossom end rot and powdery mildew of vegetable marrow in Egypt (U.A.R.). *Phytopathologia Mediterranea* **7**, 145–149.
4 GROVER R.K. (1965) A pathogenic *Cunninghamella* on pumpkin and its control. *Indian Phytopathology* **18**, 257–266.
5 HIGHAM M.T. & COLE K.M. (1982) Fine structure of sporangiole development in *Choanephora cucurbitarum* (Mucorales). *Canadian Journal of Botany* **60**, 2313–1324 illus.
6 KIRK P.M. (1984) A monograph of the *Choanephoraceae*. *Mycological Papers* No. 152, 61 pp illus.
7 MUSTAFEE T.P. (1972) Premature fruit-rot of pumpkin caused by *Choanephora cucurbitarum* in Assam. *Science and Culture* **38**, 142–143.
8 PUŞCAŞU A. (1984) [Marrow wet rot and its control.] *Producţia Vegetală, Horticultura* **33**(7), 6–8 illus.
9 SUMNER D.R. & SMITTLE D.A. (1976) Etiology and control of fruit rot of cucumber in single harvesting for pickles. *Plant Disease Reporter* **60**, 304–307.
10 WOLF F.A. (1917) A squash disease caused by *Choanephora cucurbitarum*. *Journal of Agricultural Research* **8**, 319–327 illus.

CLADOSPORIUM ROT, GUMMOSIS and **SCAB** of cucumbers, melons, watermelons, marrows, squashes and pumpkins caused by ***Cladosporium*** **spp.**

22

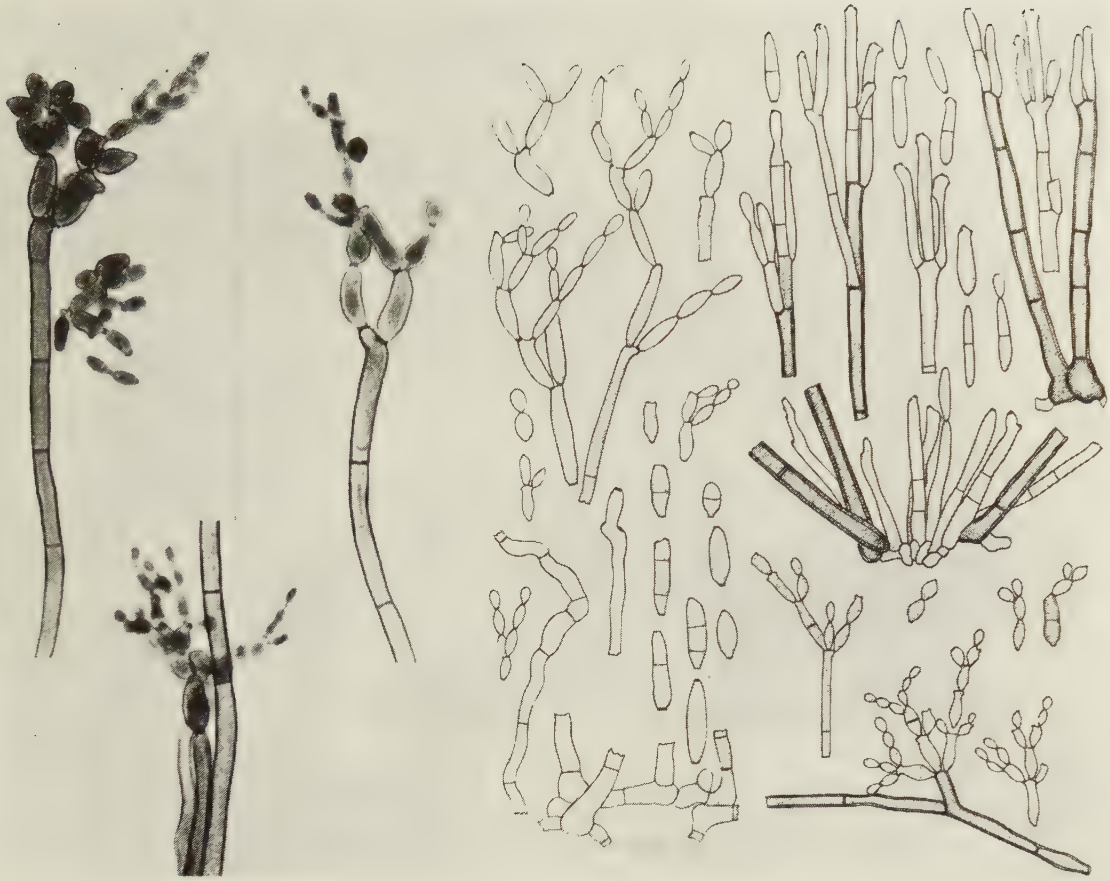


OCCURRENCE *Cladosporium cucumerinum* Ell. & Arth. causes losses mainly in temperate countries (7,8). It has been recorded on cucumbers in the northern USA (8), Canada, the Netherlands (6), Czechoslovakia (7) and the USSR (3). In Germany the disease was found to be more prevalent on outdoor cucumbers than on the greenhouse crop (2), while the reverse was found in the USA. The same fungus has also been recorded on melons and squashes in the USA (8) and on marrows in Italy (5). *C. cucumerinum* has not been recorded in Australasia but related species, *C. cladosporioides* (page 64) and *C. herbarum*, cause blemishes on Australian melons (9). *C. tenuissimum* has been recorded on watermelon fruits in India (1), and an unidentified species of *Cladosporium* has been found on Portuguese melons (4).

SYMPTOMS On immature fruits, such as young marrows, lesions caused by *C. cucumerinum* are circular (up to 1 cm in diameter), watersoaked and deeply sunken. They tend to exude a gummy substance which dries and darkens (2). Subsequently, under humid conditions, a velvety silver-grey mould develops, turning to olive-green with the formation of a dense mat of spores (conidia). On mature fruits lesions are essentially superficial and take the form of brown corky scabs. On melons symptoms vary with cultivar (10). Netted melons tend to be particularly vulnerable at the stem-scar, which may become thickly covered with dark mould; sparse mould growth may also develop between the netted areas on the fruit surface. In Honey Dews the characteristic symptom is discrete dark circular spots, each of which may be surrounded by a pale brown halo. In a humid atmosphere the smooth surface of the lesions becomes covered with olive-black mould (10). Other species of *Cladosporium* on melons may cause superficial blemishes (9), a shallow stem-end decay or severe rotting (4).

BIOLOGY These fungi survive in plant debris in the soil, and during wet or humid weather (1) they produce abundant spores (conidia) which are probably disseminated in air currents. A severe leaf blight can occur (1). *C. cucumerinum* thrives in the moist environment produced by fog or dew (7), and optimal conditions for disease development are air temperatures fluctuating around 20°C together with persistent high humidity (3,8). In cucumber crops the fungus is capable of direct penetration of young fruits, but can invade older fruits only through wounds (2,6). Incipient infections continue to develop after harvest, even under refrigeration. In one of the earliest studies of chilling injury (q.v.) in stored melons, it was noticed that cladosporium rot was often associated (10); fruit stored at too low a temperature is predisposed to general invasion by this fungus. The other species of *Cladosporium* have similar characteristics but are able to survive higher temperatures in the field.

CONTROL In cucumber an effective means of control has been the development of resistant cultivars (7). For melons, marrows, squashes and pumpkins, it may be advisable to sterilise the soil before planting, and to spray the crop with a systemic fungicide early in the growing season (5). Greenhouses should be well ventilated in order to prevent a build-up of moisture (3,6). Post-harvest rotting can be controlled by dipping the fruit in a fungicide (9), holding at the correct temperature, and avoiding prolonged storage.



23 *Cladosporium cucumerinum* (from CMI Descr. No. 348, Ellis & Holliday 1972) Conidia and conidiophores × 500

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1 AVDHESH NARAIN, SWAIN N.C., SAHOO K.C., DASH S.K. & SHUKLA V.D. (1985) A new leaf blight and fruit rot of watermelon. *Indian Phytopathology* **38**, 149–151.

2 BEHR L. (1948) Histologische Untersuchungen an krätzekranken Gurken (*Cucumis sativus* L.) unter besonderer Berücksichtigung des Krankheitsverlaufes der Krätze (*Cladosporium cucumerinum* Ell. et Arth.) an Früchten. *Phytopathologische Zeitschrift* **15**, 92–123.

3 DOROZHKIN N.A., REMNEVA Z.I. & NALOBOVA V.L. (1976) [Effects of temperature and relative humidity on the development of *Cladosporium cucumerinum* Ell. & Arth., the pathogen of green mould rot of cucumber.] *Mikologiya i Fitopatologiya* **10**, 497–503.

4 ESTEVES É. (1961) Uma doença do melão. *Agricultura* (*Revista da Direcção-Geral dos Serviços Agrícolas*) *Lisboa* **11**, 20–21 illus.

5 FRANCESCHINI A., CARTA C. & FIORI M. (1983) Prove di lotta contro il *Cladosporium cucumerinum* Ell. et Arth. agente della cladosporiosi dello zucchino. *Studi Sassaresi III* **30**, 3–9.

6 HERINGA-WESTERHOF A.C. (1946) Het Vruchtvuur in der Konkommers veroorzaakt door *Cladosporium cucumerinum* Ellis et Arthur en zijn bestrijding. *Tijdschrift over Plantenziekten* **52**, 138–139.

7 LEBEDA A. (1985) Resistance of *Cucumis sativus* cultivars to *Cladosporium cucumerinum*. *Scientia Horticulturae* **26**, 9–15.

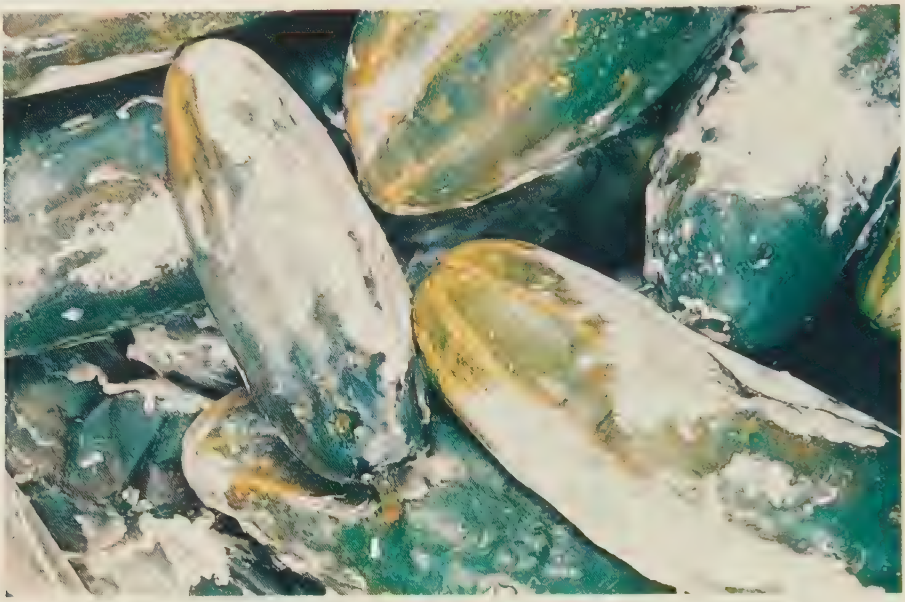
8 STRIDER D.L. & WINSTEAD N.N. (1960) Effect of temperature, pH, and various nutrients on growth of *Cladosporium cucumerinum*. *Phytopathology* **50**, 583–587.

9 WADE N.L. & MORRIS S.C. (1982) Causes and control of cantaloupe postharvest wastage in Australia. *Plant Disease* **66**, 549–552.

10 WIANT J.S. (1938) Market-storage studies of Honey Dew melons and cantaloups. *Technical Bulletin of the United States Department of Agriculture* No. 613, 18 pp illus.

COTTONY LEAK and **PYTHIUM ROT** of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes caused by ***Pythium*** spp.

24



OCCURRENCE This important disease has been recorded in many countries, both temperate and tropical. Some examples are *P. aphanidermatum* (Edson) Fitzp. on cucumbers in the USA (3,9), on cucumbers and squashes in Iraq (8), on melons in India (4) and on pumpkins in the USSR (5); *P. butleri* Subram. on cucumbers in Egypt and on melons and watermelons in India (7); *P. debaryanum* Hesse on chayotes in Italy (1); *P. myriotylum* Drechsler on watermelons in the USA (2); *P. ultimum* Trow on watermelons and pumpkins in the USA (10). Several other species of *Pythium* have been found on cucurbits (2).

SYMPTOMS Soft dark lesions develop on the fruit, often at the blossom-end or at the stem-end (10). Infected flesh undergoes a rapid breakdown, releasing copious quantities of liquid (1,10), and there may be a slight 'marshy' odour. There is usually a luxuriant growth of bright white mould, loose and fluffy (1) or densely woven (10), according to species. Numerous thick-walled spherical spores (oospores) develop in the rotted flesh. In watermelons the mould is usually confined to the internal tissues, the characteristic symptom being a dark brown lesion at the blossom-end (2).

BIOLOGY These fungi are common soil inhabitants and thrive in wet conditions. They produce both the sexual stage (oospores) and the asexual stage (sporangia which may give rise to zoospores). During rain or irrigation spores are splashed on to low-hanging fruits and, except in watermelons, can cause direct infection in the absence of wounds (10). Fruits in contact with the ground are especially vulnerable (4). Infection also occurs via the flower parts or, at harvest time, via the cut stem. The optimal growth temperatures for these fungi are in the range 28° to 37°C (3), but they can also cause severe infection

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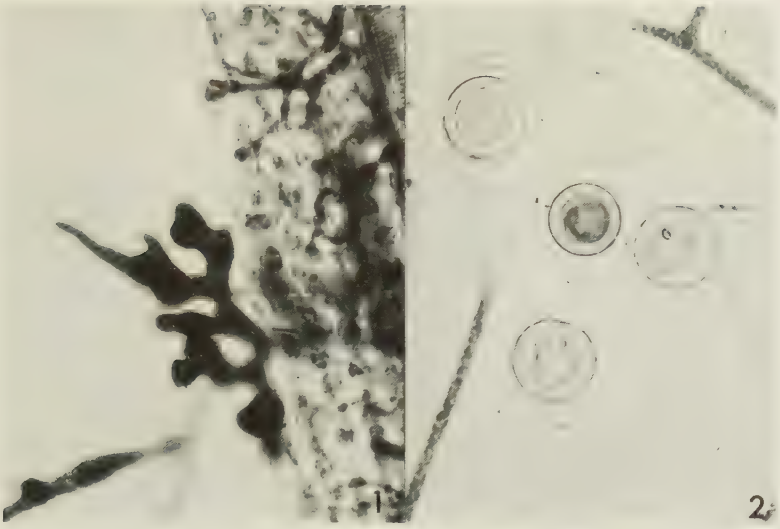

26 *Pythium aphanidermatum* (from CMI Descr. No. 36, Waterhouse & Waterston 1964) 1 sporangia × 330 2 oospores × 500 (stained with cotton blue in lactophenol)

under cool conditions (7,10). Decay spreads rapidly during transit and storage, with the formation of 'nests' of mouldy fruits exuding watery juices (1,3).

CONTROL Seed treatment is sometimes recommended, and it is advisable to practise crop rotation, excluding other susceptible crops such as tomatoes, peppers and eggplants (5). Fields should be well drained and carefully irrigated. In greenhouses a layer of plastic film on the ground serves to prevent contact between soil and fruits (4); alternatively a fungicide may be applied to the growing crop (5). Studies have been made on the use of a post-harvest dip containing antagonistic soil fungi which delay the development of pythium rot (6). During hot humid weather prompt pre-cooling of produce is essential before long distance refrigerated shipment (3).

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1 DRECHSLER C. (1925) The cottony leak of cucumbers caused by *Pythium aphanidermatum*. *Journal of Agricultural Research* **30**, 1035–1042 illus.
2 DRECHSLER C. (1939) Several species of *Pythium* causing blossom-end rot of watermelons. *Phytopathology* **29**, 391–422.
3 McCOMBS C.L. & WINSTEAD N.N. (1963) Control of cucumber cottony leak in transit. *Proceedings of the American Society for Horticultural Science* **83**, 538–546.
4 PILLAI S.N., PATEL J.R. & PATEL A.J. (1979) Control of fruit rot in watermelon. *Phytoparasitica* **7**, 17–22.
5 ROMANOVICH E.A. (1958) [New disease of pumpkins.] *Zashchita Rastenii, Moscow* **6**, 53–54.
6 SHARMA B.B., WAHAB S. & NEERAJ (1981) Studies on certain antifungal soil-fungi as control agents against cottony leak in cucurbit fruits. *Indian Phytopathology* **34**, 250–252.
7 SINGH R.S. & CHOHAN J.S. (1977) Factor affecting growth and fruit rot (cottony leak) of cucurbits caused by *Pythium butleri*. *Indian Phytopathology* **30**, 379–383.
8 TARABEIH A.M., AL-ZARARY A.J., MICHAIL S.H. & SHAWKAT A.L.B. (1979) Damping-off and fruit-rot of certain cucurbitaceous plants in Iraq. *Acta Phytopathologica Academiae Scientiarum Hungaricae* **14**, 23–30.
9 THOMPSON D.C. & JENKINS S.F. (1985) Control of cottony leak of cucumber with different formulations of metalaxyl applied at various rates and times. *Phytopathology* **75**, 1362 [Abstract].
10 TOMPKINS C.M., ARK P.A., TUCKER C.M. & MIDDLETON J.T. (1939) Soft rot of pumpkin and watermelon fruits caused by *Pythium ultimum*. *Journal of Agricultural Research* **58**, 461–475 illus.

DIDYMELLA BLACK ROT of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes caused by

Didymella bryoniae **(Auersw.) Rehm**

Conidial state: *Phoma cucurbitacearum* (Fr.) Sacc.

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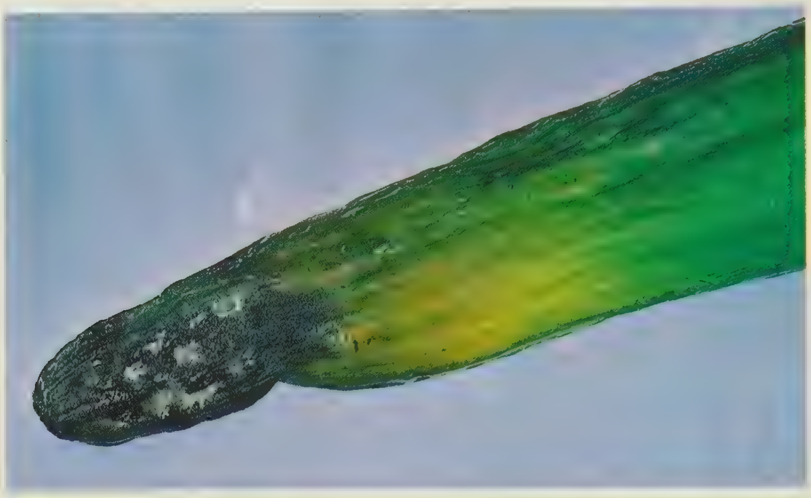
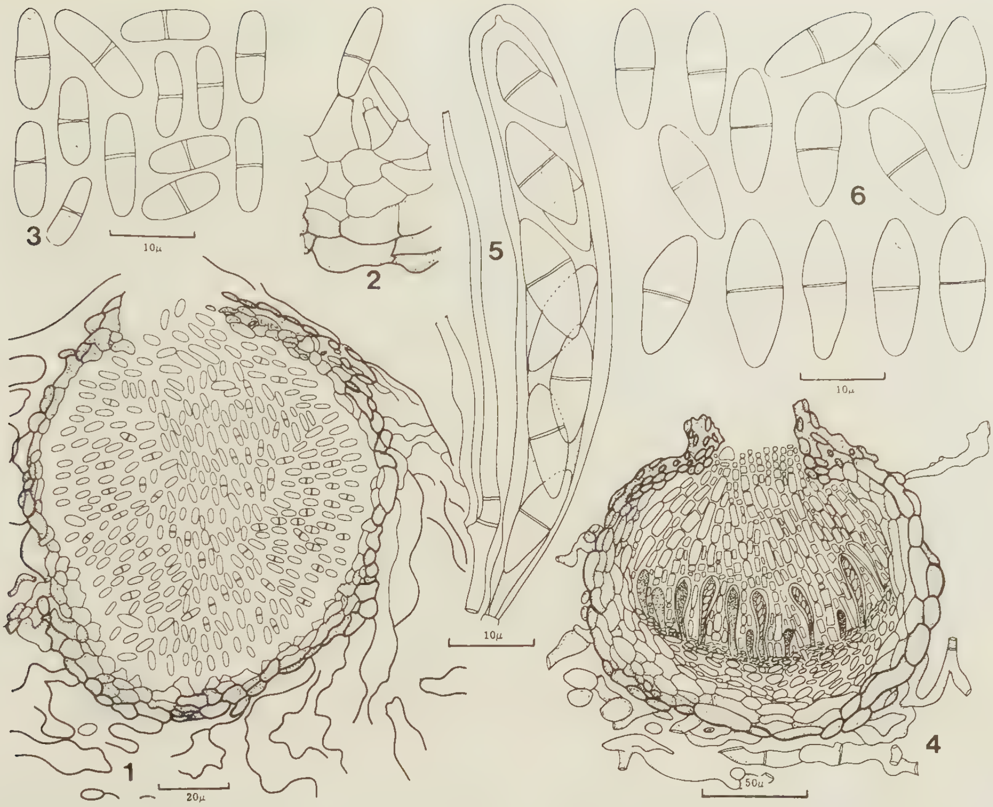


OCCURRENCE Black rot of cucurbit fruits is important both in subtropical and in cool temperate regions. For example, the disease occurs on cucumbers in the Netherlands (9) and the UK (5); on melons in Chile (7); on watermelons in the southern USA (8); on marrows in Brazil (2); on squashes in Cuba and the USA (10); on pumpkins in Trinidad (1) and Denmark (6); and on chayotes from India, Jamaica and Puerto Rico (10).

SYMPTOMS Lesions on the fruit surface are at first watersoaked and roughly circular, sometimes zonate, later becoming dark, sunken and cracked. A gummy exudate is often produced (although this is not usual in watermelons) (8) and, under humid conditions, a whitish mould may develop (10). The most characteristic symptom, however, is the production of two types of minute black bodies on the surface of the lesion, together with a gradual shrivelling and wrinkling of affected tissue. 'Internal fruit rot' (9) is characterised by a constriction of the flower end and no other obvious symptoms except a blackening of the extreme tip (10). Infected flesh is brown, soft and odourless.

BIOLOGY The fungus remains dormant in dry crop debris, and resumes activity at the onset of rain or irrigation (3). It exists, often simultaneously, in both the sexual state (perithecia giving rise to ascospores) and the asexual state (pycnidia giving rise to conidia) (7,8). The two types of spores are disseminated by wind and water respectively, and can give rise to the field disease known as gummy stem blight (1). Fruits may be invaded via the dying flowers, or through the cut stem at harvest time (2,5,9). Alternatively the fungus gains entry through injuries in the skin caused by insects during the growing season and by rough handling at harvest (8). At temperatures between 20° and 25°C rotting is rapid (3); mould strands permeate the flesh and infect the seeds, which may thereby transmit the disease to the next crop (6,7).

CONTROL Debris from any previous infected crop should be promptly removed and destroyed (7). Clean or treated seed should be used, and in high value protected crops it may be advisable to carry out soil sterilisation before planting. Insects should be controlled during the growing season, and fungicide sprays applied (4). Irrigation should be frequent so as to prevent drought stress (9). Where flower infection is common it has been recommended that dead flowers be removed by hand during periods when temperature and humidity are conducive to infection (2). Beneficial reduction of humidity in greenhouses can be achieved by judicious ventilation (5) and the use of supplementary heating early in the mornings. Care is needed at harvest time, firstly to ensure that the fungus is not transmitted to the cut stem by contaminated knives (5) and secondly to prevent unnecessary injury to the skin of the fruit (8). If infection has occurred, decay can be partially controlled by prompt cooling and storage at the optimal temperature for the cultivar concerned (4). A few cultivars of cucumber, melon and watermelon are resistant, some by virtue of their flowering characteristics (9).



28 *Didymella bryoniae* (from CMI Descr. No. 332, Punithalingam & Holliday 1972) 1 pycnidium 2 part of pycnidial wall and conidiophores 3 conidia 4 pseudothecium 5 ascus and pseudoparaphysis 6 ascospores

1 BALA G. & HOSEIN F. (1986) Studies on gummy stem blight disease of cucurbits in Trinidad. *Tropical Agriculture* **63**, 195–197.

2 CARDOSO R.M.G., FIGUEIREDO M.B., PALAZZO D. & MARTINEZ J.A. (1974) Epidemiology of fruit rot (*Mycosphaerella melonis* (Pass.) Chiu & J.C. Walker) on Italian squash (*Cucurbita pepo* L.). *Arquivos do Instituto Biológico* **41**, 35–37.

3 CHIU W.F. & WALKER J.C. (1949) Physiology and pathogenicity of the cucurbit black rot fungus. *Journal of Agricultural Research* **78**, 589–615.

4 DOW A.T., SEGALL R.H., HOPKINS D.L. & ELSTROM G.W. (1978) Effects of storage temperature and field fungicide treatments on decay of Florida watermelons. *Proceedings of Florida State Horticultural Society* **91**, 149–150.

5 FLETCHER J.T. & PREECE T.F. (1967) Mycosphaerella stem rot of cucumbers in the Lea Valley. *Annals of Applied Biology* **58**, 423–430.

6 LEE D.-H., MATHUR S.B. & NEERGAARD P. (1984) Detection and location of seed-borne inoculum of *Didymella bryoniae* and its transmission in seedlings of cucumber and pumpkin. *Phytopathologische Zeitschrift* **109**, 301–308.

7 SANZ B.-M., H. (1971) *Mycosphaerella melonis* (Pass.) Chiu and Walker, una nueva enfermedad de las cucurbitáceas en el país. *Agricultura Técnica* **31**, 142–144.

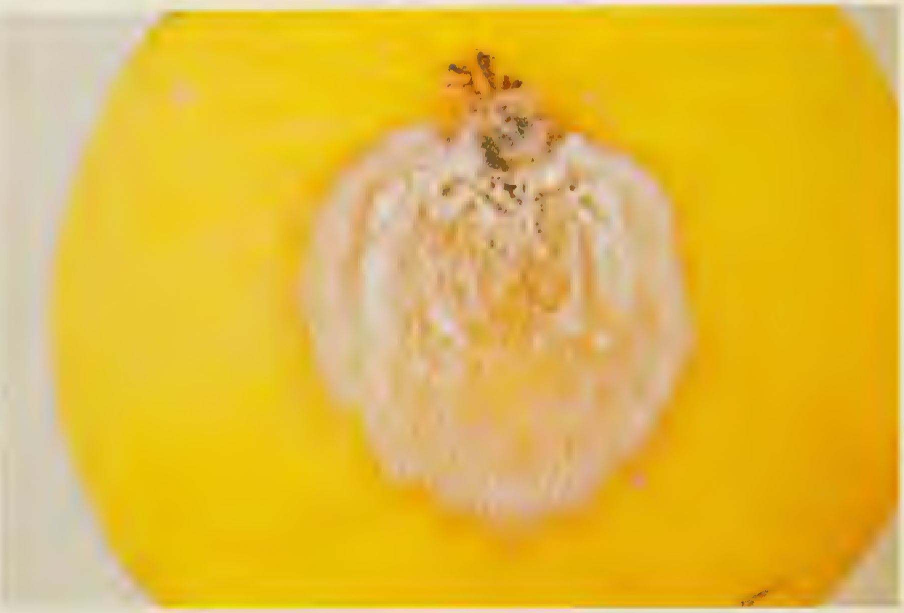
8 SCHENCK N.C. (1962) Mycosphaerella fruit rot of watermelon. *Phytopathology* **52**, 635–638 illus.

9 STEEKELENBURG N.A.M. van (1986) Factors influencing internal fruit rot of cucumber caused by *Didymella bryoniae*. *Netherlands Journal of Plant Pathology* **92**, 81–91 illus.

10 WIANT J.S. (1945) Mycosphaerella black rot of cucurbits. *Journal of Agricultural Research* **71**, 193–213 illus.

FUSARIUM ROT of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes caused by ***Fusarium*** **spp.**

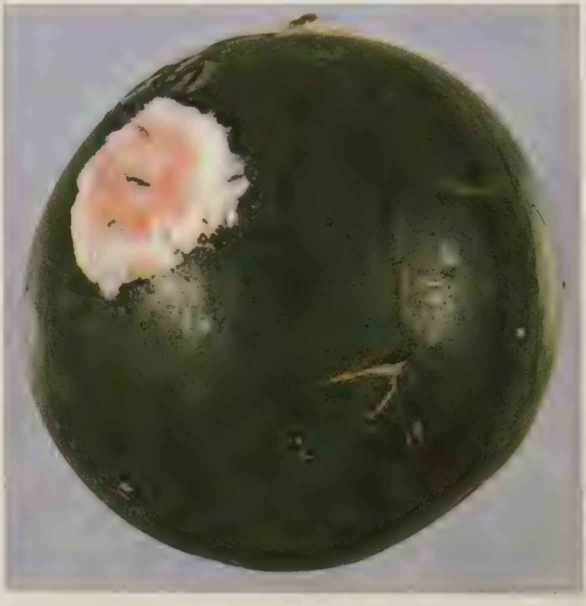
29



OCCURRENCE Several species of *Fusarium* have been found on cucurbit fruits. Examples are *F. equiseti* (Corda) Sacc. on melon in Turkey (3) and on watermelon in India (8); *F. oxysporum* on cucumber and watermelon in the USA (4,6); *F. pallidoroseum* (Cooke) Sacc. on melon and squash in France (9), and on melon in India (10), central America and the USA (1); and *F. solani* (Mart.) Sacc. on cucumber and melon in Israel (5), on watermelon in Nigeria (2) and on squash in the Netherlands (7). Fusarium rot of chayotes has been reported from India.

SYMPTOMS Lesions may occur anywhere on the fruit but are frequently found at the stem-end (9). Affected tissue tends to be spongy or corky and, under humid conditions, becomes covered with a white or pinkish mould. Decay may be shallow or extend to the seed cavity, and there is usually a distinct line of demarcation between healthy and rotted tissue (1,9). A reddish-purple pigment is sometimes produced (cf. charcoal rot, q.v.).

BIOLOGY These fungi are common in soil, and survive as thick-walled resting spores (chlamydospores). These germinate to produce mould strands which can invade plant roots and cause wilting, followed by stem-rot and fruit-rot (4). Thin-walled asexual spores (conidia) are also produced, and these are disseminated by wind and water. A wound is necessary for infection through the skin of the fruit (3), and may be in the form of insect damage (2) or abrasion; disease incidence is especially high in thin-skinned cultivars (10). Further infections, resulting in a stem-end rot, can be caused during harvesting if knives become contaminated through contact with soil or with infected plant tissue. The optimal growth temperatures for these fungi lie between 22° and 29°C. If decay progresses to the seed cavity, the seeds become infected and may transmit the disease to the next crop.



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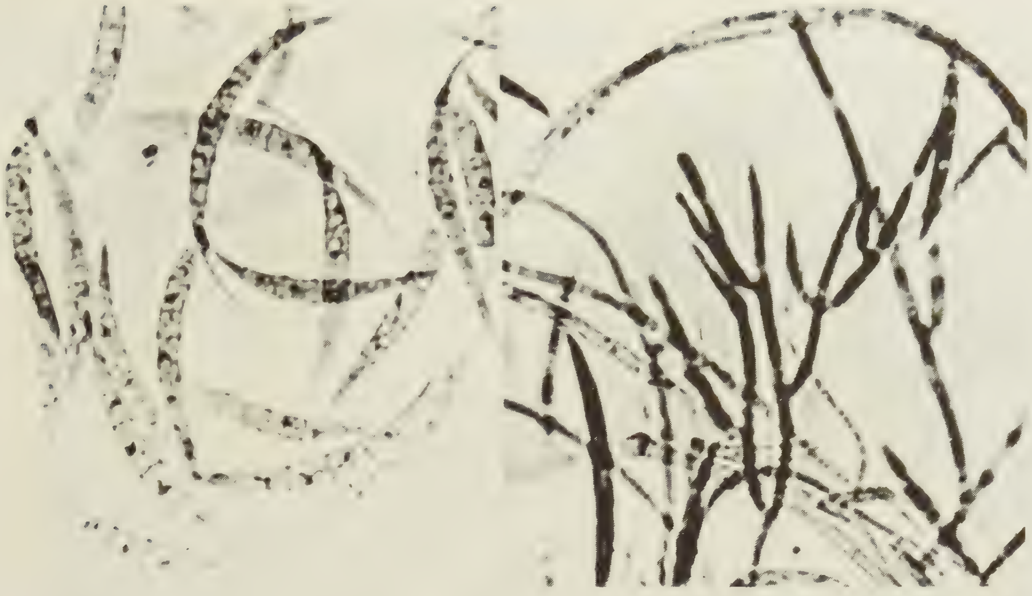


31 *Fusarium equiseti* (from CMI Descr. No. 571, Booth 1978) Conidia and conidiophores × 750 (stained with cotton blue in lactophenol)

CONTROL It may be necessary to disinfect the soil (7), to use a seed dressing, and to protect the developing crop from insect pests (2). A systemic fungicide can be sprayed on to the growing crop, and its protective action continues after harvest (9). Careful inspection in the packhouse (1) helps to eliminate most fruits with slight pre-harvest infections, and gentle handling serves to minimise the incidence of wound infections (3). Post-harvest treatments alone are not always effective (9), although improved control can be achieved by heating the fungicide dip. Subsequently fruit should be held under refrigeration, since at 10°C these fungi develop slowly.

1 CARTER W.W. (1979) Corky dry rot of cantaloup caused by *Fusarium roseum* 'semitectum'. *Plant Disease Reporter* **63**, 1080–1084 illus.

2 IKEDIUGWU F.E.O. & OGIEVA W.O. (1978) Fruit rot of *Citrullus lanatus* in Nigeria caused by *Fusarium solani. Transactions of the British Mycological Society* **71**, 209–213.

3 IREN S. & SORAN H. (1973) Untersuchungen über die Feststellung und pathogenität des Zuckersmelonenfruchtfäuleerregers *Fusarium equiseti* (Corda) Sacc. *Journal of Turkish Phytopathology* **2**, 130–139.

4 JENKINS S.F. & WEHNER T.C. (1983) Occurrence of *Fusarium oxysporum* f.sp. *cucumerinum* on greenhouse-grown *Cucumis sativus* seed stocks in North Carolina. *Plant Disease* **67**, 1024–1025.

5 JOFFE A.Z. & PALTI J. (1972) *Fusarium* species of the *Martiella* section in Israel. *Phytopathologische Zeitschrift* **73**, 123–148.

6 McMILLAN R.T. (1986) Cross pathogenicity studies with isolates of *Fusarium oxysporum* from either cucumber or watermelon pathogenic to both crop species. *Annals of Applied Biology* **109**, 101–105.

7 PATERNOTTE S.J. (1987) Pathogenicity of *Fusarium solani* f.sp. *cucurbitae* race 1 to courgette. *Netherlands Journal of Plant Pathology* **93**, 245–252.

8 SUMBALI G. & MEHROTRA R.S. (1982) Cucurbitaceous fruits – new hosts for *Fusarium equiseti. National Academy of Science Letters* **5**, 121–122.

9 TROUILLON P. & TISSUT M. (1972) Contribution à l'étude d'une fusariose de fruits de cucurbitacées et de ses possibilités de traitement. *Phytiatrie-Phytopharmacie* **21**, 61–67.

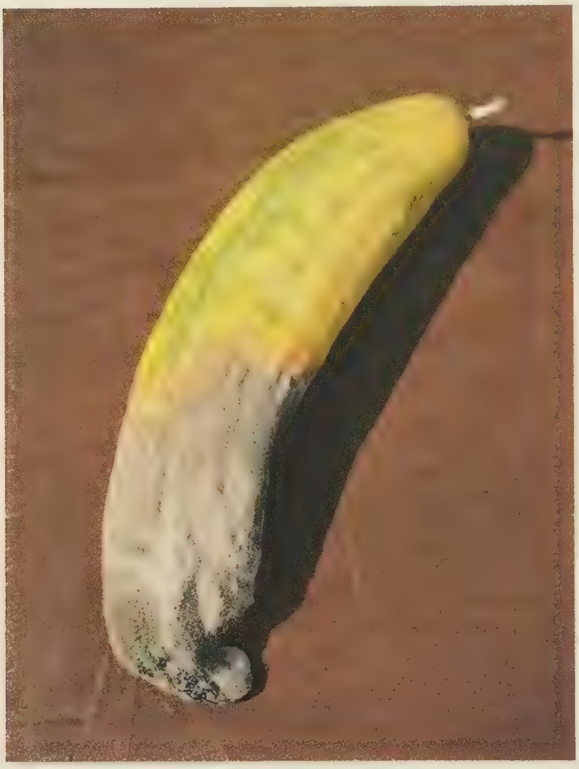
10 WARAITCH K.S. & NANDPURI K.S. (1975) Fusarium fruit rot of muskmelon (*Cucumis melo* L.). *Journal of Research of Punjab Agricultural University* **12**, 131–134.

GREY MOULD ROT of cucumbers, melons, marrows, squashes and pumpkins caused by

Botryotinia fuckeliana **(de Bary) Whetzel**

Conidial state: *Botrytis cinerea* Pers.

32



OCCURRENCE Grey mould rot is especially important on cucumbers in cool temperate countries, for example Japan (2), the northern USA (3), Canada (4) and the UK. It has caused losses of melons in France (1) and squashes in Israel (5); marrows and pumpkins may also be attacked.

SYMPTOMS The infected area (frequently at the flower end) is soft, watersoaked and yellowish, later becoming covered with abundant grey mould, spores (conidia) and occasionally irregular shaped black resting bodies (sclerotia) approximately 2 to 3 mm across.

BIOLOGY The fungus survives on plant debris in the soil and, during wet weather or humid greenhouse conditions, it produces abundant asexual spores (conidia) which are disseminated in air currents (2). In the presence of moisture dying blossoms are colonised by the mould, which is subsequently able to invade the fruit (3,4). Infection may also occur via wounds or through the cut stem at harvest time. The optimal growth temperature for the fungus is approximately 20°C, but substantial rotting can also occur at normal cold store temperatures for cucurbits (pages 12–15).

CONTROL Strict hygiene is important; prompt destruction of crop debris helps to reduce the number of spores released into the atmosphere. Spore production can be inhibited in greenhouses made from special plastic film which cuts out certain ultraviolet wavelengths (2); however, inhibition may be short-lived as the fungus adapts. Irrigation and ventilation should be well controlled to prevent the build-up of excessive moisture, and fungicide sprays should be carefully timed (4). Some fungicides remain effective for only one or two seasons, owing to the emergence of new strains of fungus tolerant of the chemical. Control measures are similar to those for *Sclerotinia* species (see watery soft rot) (1). The two diseases often occur in the same crop (3), though one or the other may predominate at different times of year (5). If incipient infections are suspected, fruits can be treated with a fungicide immediately after harvest (5). Refrigeration can retard rotting but does not stop it.

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1 ANON. (1979) Lutte contre les pourritures des fruits de cucurbitacées (*Botrytis cinerea–Sclerotinia sclerotiorum*). *Revue Horticole* No. 200, 35.

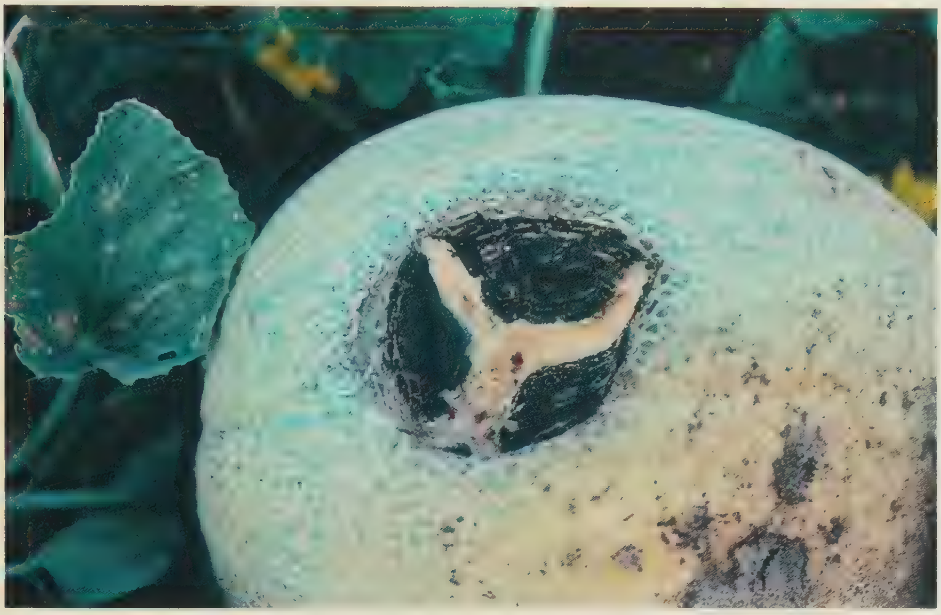
2 HONDA Y. & YUNOKI T. (1980) Inhibition of fungal sporulation by ultraviolet-absorbing vinyl film and its application to disease control. *Japan Agricultural Research Quarterly* **14**, 78–83.

3 KADOW K.J., ANDERSON H.W. & HOPPERSTEAD S.L. (1938) Control of *Sclerotinia* and *Botrytis* stem rots of greenhouse tomatoes and cucumbers. *Phytopathology* **28**, 224–227 illus.

4 McKEEN C.D. (1952) Observations on the occurrence and control of grey mould on greenhouse cucumbers. *Scientific Agriculture* **32**, 670–676.

5 TEMKIN-GORODEISKI N. (1970) Storage rots of zucchini squash (*Cucurbita pepo*) and their control. *Israel Journal of Agricultural Research* **20**, 97–99.

MYROTHECIUM ROT of cucumbers, melons and watermelons caused by ***Myrothecium roridum*** Tode ex Fr.



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OCCURRENCE This fungus has been recorded on cucumbers (5), melons and watermelons in India and on melons from the southern USA and Mexico (1,2,3). A related species, *M. verrucaria* Ditm. ex Fr., is reported on cucumbers in the USSR (4).

SYMPTOMS Lesions on cucumbers are irregular, slightly depressed, and turn from greenish-yellow to dirty red (5); on melons they are dark and sunken. Affected areas support discrete greenish-black spore-masses (sporodochia) which are 1 to 1.5 mm in diameter and fringed with white (3). These bodies may coalesce into a sticky charcoal-black sheath, or become dry, hard and flaky. There is a gradual rotting of the flesh, which becomes watery but does not have a pronounced odour (3). Under humid conditions there is a luxuriant growth of whitish mould, interspersed with spore-masses (5).

BIOLOGY The disease may be seed-borne (4). The fungus is also common in soil, and its spores (conidia) are probably disseminated by wind, water and insects. During warm wet weather infection causes leaf-spotting on many crops; furthermore, flowers may become infected and symptoms are later manifest on fruits in field and packhouse (1). Rotting occurs at temperatures between 10° and 30°C (5). Cultivars differ substantially in their tolerance of this disease (2).

CONTROL Seed treatment may be advisable (4). Field hygiene is important; diseased plant debris should be removed and destroyed before the new crop is planted. A fungicide spray can be effective if applied at the onset of flowering (1). If infected fruit is inadvertently packed, deterioration can be delayed by the use of refrigeration.

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1 CARTER W.W. (1980) Incidence and control of *Myrothecium roridum* on cantaloup in relation to time of fungicide application. *Plant Disease* **64**, 872–874.

2 KUTI J.O., NG T.J. & BEAN G.A. (1987) Reactions of muskmelon cultigens to *Myrothecium roridum*. *HortScience* **22**, 635–637.

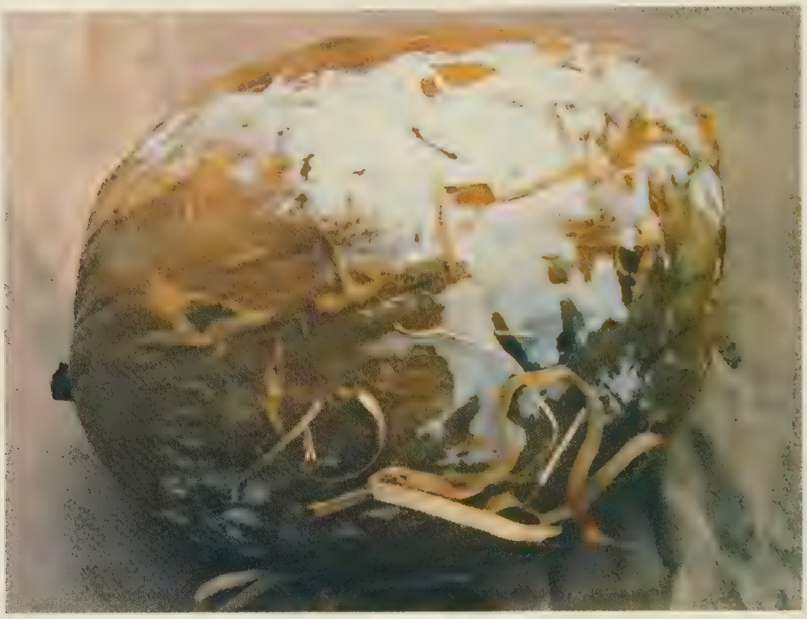
3 McLEAN D.M. & SLEETH B. (1961) *Myrothecium* rind rot of cantaloup. *Plant Disease Reporter* **45**, 728–729 illus.

4 NIKOLAEVA S.I. & MARZHINA L.A. (1982) [*Myrothecium verrucaria* Ditm. ex Fr., a new pathogen of mycosis of cucumber.] *Mikologiya i Fitopatologiya* **16**, 358–359.

5 RAO V.G. & SUBRAMONIAM V. (1974) Studies into *Myrothecium* rot of cucumber. *Rivista di Patologia Vegetale, Padova IV* **10**, 411–426.

PHYTOPHTHORA ROT of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes caused by ***Phytophthora* spp.**

34



OCCURRENCE Several species of *Phytophthora* cause rotting in cucurbit fruits. Examples are *P. cactorum* (Leb. & Cohn) Schroet. on watermelons in the USA (2); *P. capsici* Leonian on melons in the USA (8), on squashes in Italy (6) and in Argentina (7) and on pumpkins in Japan (4); *P. drechsleri* Tucker on cucumbers and melons in China and on marrows in Egypt (3); and *P. nicotianae* var. *parasitica* on watermelons in the USA (5) and in India (9) and on chayotes in Mauritius (1). Phytophthora rot has also been recorded on Chilean melons imported into the USA (10).

SYMPTOMS Lesions may form on any part of the fruit, and are usually brown with an irregular margin, later becoming zoned or mottled. Under humid conditions a whitish mould develops, on which are produced characteristic spore-heads (sporangia). The skin may rupture, and eventually the entire fruit decays. Infected flesh is soft, watersoaked, rarely discoloured, and almost odourless unless invaded by secondary organisms (2,7,8,9,10).

BIOLOGY These fungi are common soil inhabitants, causing disease under warm wet conditions. They produce both the sexual stage (oospores) (4) and the asexual stage (sporangia which may give rise to zoospores), and infection is often from zoospores disseminated by rain or irrigation water (1,5). Fruits on or near the ground are most likely to be invaded and, in the presence of moisture, the fungus can penetrate the unbroken skin (6,8,9). Fruits may be harvested in apparently good condition, and undergo decay during transport and storage; the mould

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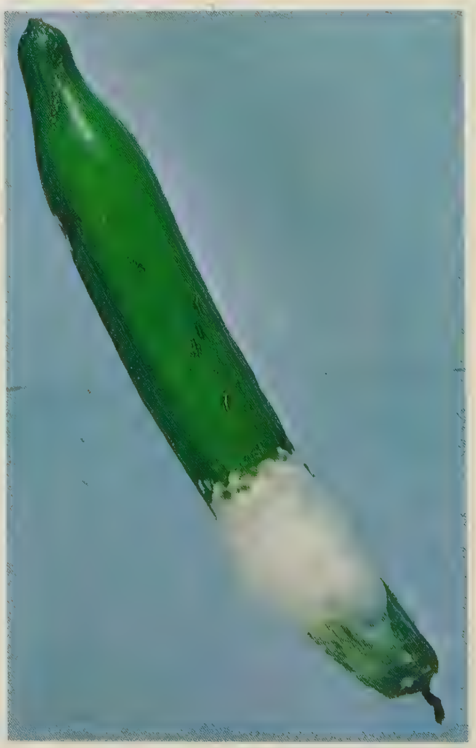

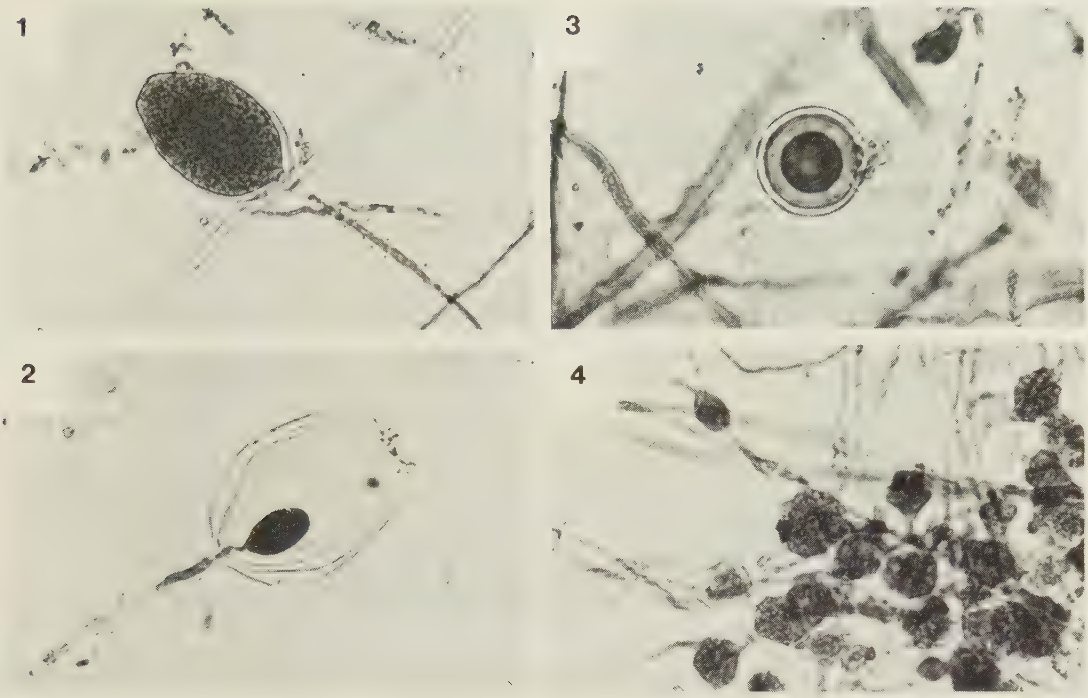

36 *Phytophthora drechsleri* (from CMI Descr. No. 840, Stamps 1985) 1 sporangium 2 sporangium showing internal proliferation 3 oospore 4 hyphal swellings × 500 (stained with cotton blue in lactophenol)

spreads from infected fruits into adjacent sound fruits (10). The optimal growth temperatures for these species of *Phytophthora* lie between 25° and 30°C, and rotting is slow at 10°C and below (3,6,8,10).

CONTROL Fields should be well drained, and properly irrigated so that water does not accumulate (8). A soil fungicide may be applied (6). Decay in harvested fruits can be minimised by the use of refrigeration.

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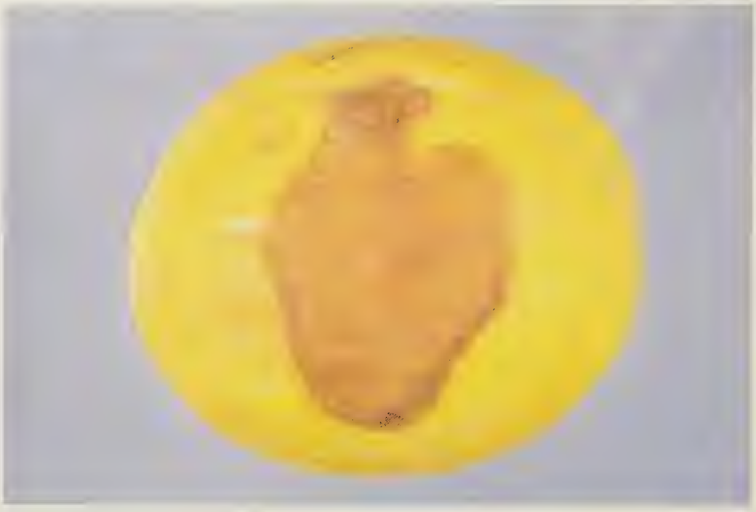
1 ANON. (1963) Plant pathology. *Report of the Department of Agriculture, Mauritius, 1962*, 43–47.
2 BROWN J.G. & EVANS M.M. (1933) A *Phytophthora* rot of watermelon. *Technical Bulletin of Arizona Agricultural Experiment Station* No. 51, 45–65 illus.
3 EL-HELALY A.F., ASSAWAH M.W., ELAROSI H.M. & WASFY E.-E.H. (1968) Fruit rots of vegetable marrow in Egypt (United Arab Republic). *Phytopathologia Mediterranea* **7**(2–3), 107–115 illus.
4 KAMJAIPAI W. & UI T. (1978) Mating types of *Phytophthora capsici* Leonian, the causal fungus of pumpkin rot in Hokkaido. *Annals of the Phytopathological Society of Japan* **44**, 440–446.
5 NORTON D.C. & ROSBERG D.W. (1954) Watermelon fruit rot, caused by *Phytophthora parasitica*, in Texas. *Plant Disease Reporter* **38**, 854 illus.
6 NOVIELLO C., CRISTINZIO G. & ALOJ B. (1977) Una grave malattia della zucca in Campania. *Annali della Facoltà di Scienze Agrarie della Università degli Studi di Napoli Portici* **11**, 11–22 illus.
7 PONTIS R.E. (1945) *Phytophthora capsici* en frutos de zapallito de tronco. *Revista Argentina de Agronomía* **12**, 17–21 illus.
8 TOMPKINS C.M. & TUCKER C.M. (1937) *Phytophthora* rot of honeydew melon. *Journal of Agricultural Research* **54**, 933–944 illus.
9 ULLASA B.A. (1984) Two post-harvest diseases of water-melon caused by *Phytophthora nicotianae* and *Fusarium oxysporum*. *FAO Plant Protection Bulletin* **32**, 145 illus.
10 WIANT J.S. & TUCKER C.M. (1940) A rot of Winter Queen watermelons caused by *Phytophthora capsici*. *Journal of Agricultural Research* **60**, 73–88 illus.

PINK MOULD ROT of melons and watermelons caused by *Trichothecium roseum* Link

37



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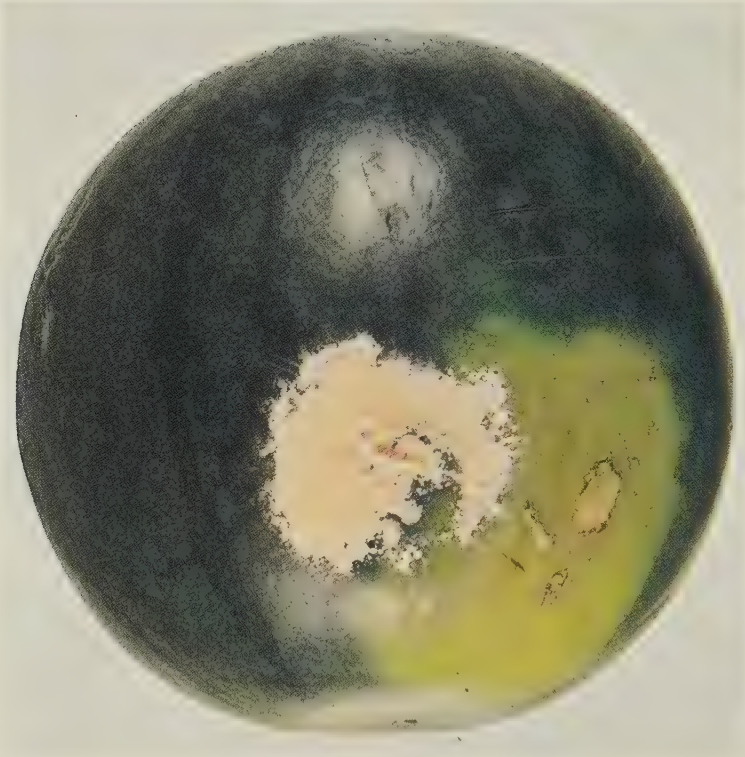
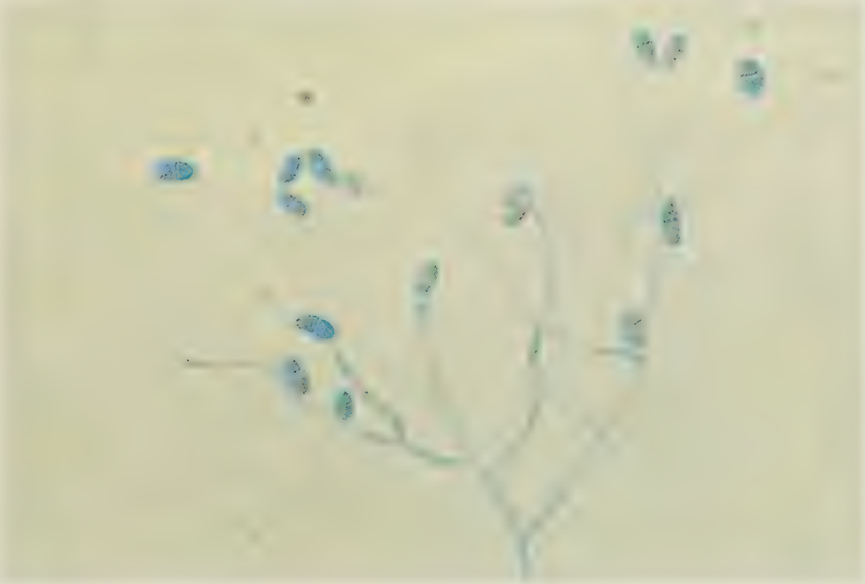


OCCURRENCE Pink mould rot has been recorded on melons and watermelons in India (8,9), on melons in the USA (10), and on melons imported from South America (10). The author has observed the disease on melons imported into the UK from Spain and Chile. The causal fungus also occurs on melons in France (1) and in Israel (2).

SYMPTOMS Lesions form at the blossom-end, stem-end or side of the fruit, and may extend over much of the surface. Affected skin becomes somewhat tough and shrivelled, and a viscous liquid may ooze from the lesion. Diseased flesh is spongy, slightly brown and extremely bitter in taste. Under humid conditions there is profuse development of salmon-pink spores (3,5,8).



39 Conidia of *Trichothecium roseum* (stained with Trypan blue)

BIOLOGY The fungus is a common soil inhabitant, and its spores (conidia) are probably dispersed by air currents, irrigation water and insects. It can infect stems, leaves and flowers of cucumber plants weakened by low night temperatures, poor lighting and ventilation, and excessive humidity (4,5); however, there are no records of its occurrence on cucumber fruits. Melon and watermelon fruits are invaded via the blossom-end, through injuries, or at the cut stem at harvest time. The optimal growth temperature for the fungus lies between 20° and 25°C, and decay is very slow at temperatures below 10°C.

CONTROL Field hygiene is important; for example the fungus may be inadvertently introduced in mulching materials (6). Care should be taken to maintain the plants in a vigorous condition, and to prevent insect injury or mechanical damage. Fungicide sprays applied to the growing crop provide partial control of post-harvest decay (1). Deterioration can be further delayed by the use of post-harvest chemical treatments (9) and refrigerated storage. Pink mould is extremely sensitive to low-dose γ-irradiation.

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1 ANON. (1979) Lutte contre les pourritures des fruits de cucurbitacées (*Botrytis cinerea-Sclerotinia sclerotiorum*). *Revue Horticole* No. 200, 35.
2 BARKAI-GOLAN R., KARADAVID R. & BALASHA O. (1979) The role of pectolytic and cellulolytic enzymes in *Trichothecium* and *Fusarium* rots in melon. *Phytoparasitica* **7**, 43 [Abstract].
3 INGOLD C.T. (1956) The conidial apparatus of *Trichothecium roseum. Transactions of the British Mycological Society* **39**, 460–464.
4 KALASHNIKOFF K.J. (1935) [*Trichothecium roseum* Link on cucumber plants under glass.] *Plant Protection, Leningrad* **7**, 136–139.
5 KENDRICK W.B. & COLE G.T. (1969) Conidium ontogeny in Hyphomycetes. *Trichothecium roseum* and its meristem arthrospores. *Canadian Journal of Botany* **47**, 345–350 illus.
6 McKEEN C.D. (1954) *Trichothecium* foliage rot of greenhouse cucumbers. *Canadian Journal of Agricultural Science* **34**, 469–472.
7 MITIDIERI I.M. de (1973) Enfermedades criptogámicas nuevas o poco difundidas en la Argentina. *IDIA* No. 301, 9–12 illus.
8 RAO V.G. (1964) Some new market and storage diseases of fruits from India. *Plant Disease Reporter* **48**, 629–633 illus.
9 SINGH J.-P. & BHATNAGAR D.K. (1982) Controlling post-harvest decay of muskmelon fruits by coating wax-emulsion and fungicides. *Haryana Agricultural University Journal of Research* **12**, 295–297.
10 WIANT J.S. (1937) Investigations of the market diseases of cantaloups and Honey Dew and Honey Ball melons. *Technical Bulletin of the United States Department of Agriculture* No. 573, 47 pp illus.

RHIZOPUS ROT of cucumbers, melons, watermelons, marrows, squashes and pumpkins caused by

Rhizopus oryzae **Went & Prinsen Geerligs**
Rhizopus stolonifer **(Ehrenb. ex Fr.) Lind**

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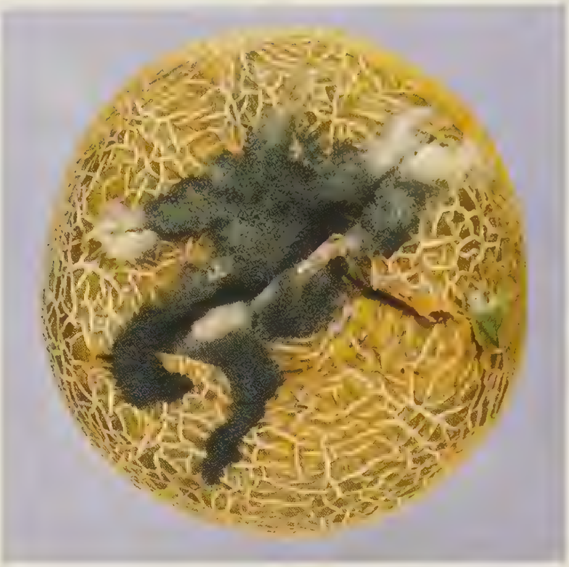

OCCURRENCE Rhizopus rot is of particular importance on melons, for example in Israel (1), Japan (3), Australia (5), South Africa, the USA and Chile. The disease has also caused losses of cucumbers and squashes in the USA (2,4), marrows and pumpkins in Israel (1) and watermelons in India.

SYMPTOMS Lesions are soft and watersoaked, with a fairly distinct boundary. The skin often cracks open, and coarse white mould strands develop on the surface, later giving rise to a mass of globular spore-heads (sporangia) which turn from white to black (3). Diseased flesh is soft, very wet, and usually sharply delimited from healthy tissue, but it is held together by strands of mould and so does not disintegrate completely. A faintly sour odour may accompany the decay.

BIOLOGY These moulds exist in plant debris in the soil. They occasionally produce the sexual state (zygospores) but much more common is the asexual state (sporangia giving rise to sporangiospores), these spores being blown around in the atmosphere. During wet weather dying blossoms may be invaded, and the fungus thereby gains entry into the fruit (2,4). Alternatively, infection of mature fruits can occur through wounds (5). Ripe melons are especially susceptible. Optimal growth temperatures are 35°C for *R. oryzae* and 25°C for *R. stolonifer.* Decay is very slow at 10°C and below (2).

CONTROL It may be necessary to apply a fungicide spray to the growing crop (4). Care should be taken to prevent injury to the fruits during harvesting and handling; refrigerated storage is effective in checking decay (5).

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1 BARKAI-GOLAN R. (1981) An annotated check-list of fungi causing postharvest diseases of fruits and vegetables in Israel. *Special Publication of the Volcani Center* No. 194, 36 pp.

2 NEWHALL A.G. & WILKINSON R.E. (1949) Storage rots of squash in New York state. *Plant Disease Reporter* **33**, 220.

3 SHIDA T., FURUKI I., ARIMOTO Y., HOMMA Y., TUBAKI K. & MISATO T. (1982) [Occurrence of muskmelon black mold caused by *Rhizopus stolonifer.*] *Annals of the Phytopathological Society of Japan* **48**, 702–704.

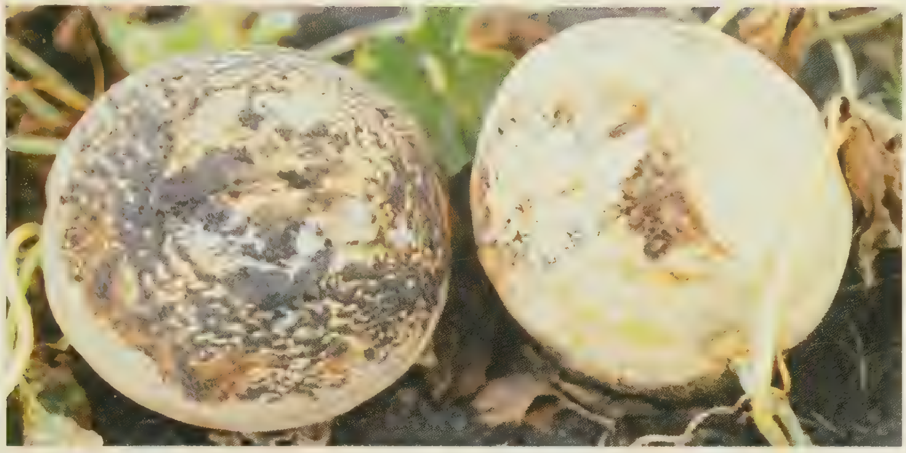
4 SONODA R.M. (1980) Rhizopus soft rot of seedless greenhouse cucumbers. *Plant Disease* **64**, 296–297 illus.

5 WADE N.L. & MORRIS S.C. (1982) Causes and control of cantaloupe post-harvest wastage in Australia. *Plant Disease* **66**, 549–552.

SCLEROTIUM ROT of melons, watermelons, squashes and pumpkins caused by

Corticium rolfsii **Curzi**

Sclerotial state: *Sclerotium rolfsii* Sacc.



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OCCURRENCE Sclerotium rot occasionally causes losses in fruits grown under warm wet conditions. The disease has been recorded on melons in Israel, on melons and watermelons in the southern USA (3,4,5), on watermelons in Brunei, Nigeria (2) and Mexico (4), on squashes in southern Italy (1) and on pumpkins in India.

SYMPTOMS Lesions are soft, watery and discoloured, and may exude amber droplets (3,5). The skin of the fruit often cracks open, and a fine white silky mould spreads over the surface, characteristically flat and fan-like in appearance (4). Spherical bodies (sclerotia) are produced, approximately 1 mm in diameter, white at first and later dark brown. The mould can grow on paper used for packaging (5).

BIOLOGY The fungus persists in the soil as resting bodies (sclerotia) which, under warm wet conditions, give rise to strands of mould. Fruits in contact with the ground can be invaded (5), especially if the skin has been punctured by insects (2) or has suffered mechanical damage (4). Epidemics have occurred after heavy rains (5). Incipient infections may not be noticed at harvest time, and can lead to serious rotting during transport and storage (4,5). Decay is very rapid at temperatures above 20°C, the optimal growth temperature for the fungus being approximately 30°C (3). Invasion of the seed makes it useless for consumption or for planting (2).

CONTROL It is not easy to rid the soil of this fungus, but partial control may be achieved with chemicals or by means of solarization. In the latter method a plastic tarpaulin is spread on the field for several weeks before planting; during hot dry weather the resulting soil temperatures can be lethal to the fungus. An alternative strategy is to prevent fruits from coming into contact with infective soil by using a mulch or by raising them off the ground with an arrangement of sticks. Fungicide treatment of harvested fruits has been recommended, and cool storage holds decay in check (3).

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1 FRANCESCHINI A., CARTA C. & FIORI M. (1982) Il "mal dello sclerozio" dello zucchino (*Cucurbita pepo* L.) da *Sclerotium rolfsii* Sacc. in Sardegna. *Studi Sassaresi III* **29**, 423–429.

2 IKEDIUGWU F.E.O. (1980) *Corticium rolfsii* and fruit rot of *Citrullus lanatus* in the field in Nigeria. *Transactions of the British Mycological Society* **75**, 316–319.

3 LUEPSCHEN N.S. (1961) The development of *Mycosphaerella* black rot and *Pellicularia rolfsii* rot of watermelons at various temperatures. *Plant Disease Reporter* **45**, 557–559 illus.

4 RAMSEY G.B., SMITH M.A., BERAHA L. & WRIGHT W.R. (1959) *Pellicularia rolfsii* on Mexican and Texas watermelons. *Plant Disease Reporter* **43**, 1031 illus.

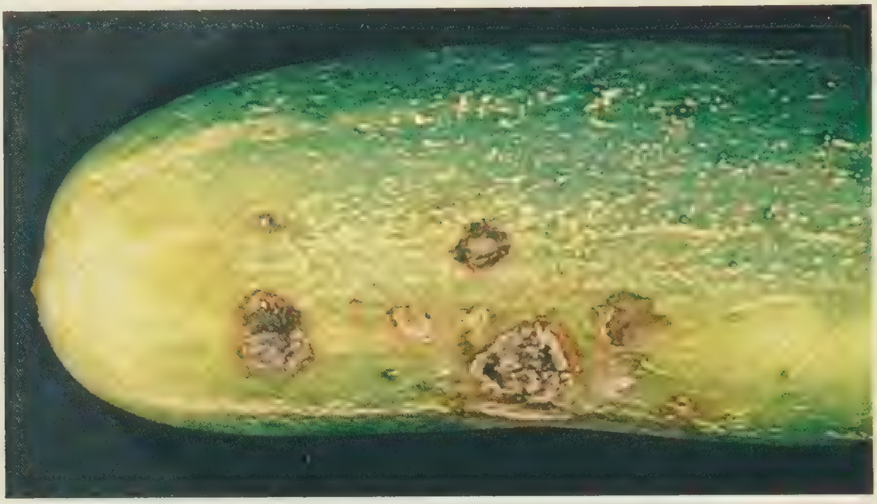
5 ROSEN H.R. & SHAW L. (1929) Studies on *Sclerotium rolfsii*, with special reference to the metabolic interchange between soil inhabitants. *Journal of Agricultural Research* **39**, 41–61 illus.

SOIL ROT of cucumbers, melons, watermelons and squashes caused by

***Thanatephorus cucumeris* (Frank) Donk**

Sclerotial state: *Rhizoctonia solani* Kühn

42



OCCURRENCE This disease occurs in warm wet growing areas. It is occasionally found on melons and watermelons but is more important on cucumbers, for example in the southern USA (2) and parts of the USSR. Squashes are attacked in Egypt (3) and melons in India (4). A spongy rot of pumpkin in India is reported to be caused by a disease complex including soil rot (1).

SYMPTOMS Soil rot lesions are at first firm, brownish and watersoaked, enlarging into irregular-shaped sunken cankers. In a warm humid atmosphere rotting is rapid, and infected fruits become covered with a dense greyish-brown mould (2).

BIOLOGY The fungus is common in soil, and is capable of attacking a wide range of crops. It may produce both the sexual state (a hymenium giving rise to basidiospores) and the asexual state (crust-like sclerotia), and can survive on weeds, in crop residues or as sclerotia in the soil (2). Under wet conditions the mould can attack fruits in contact with the ground; rotting may not be apparent until after harvest. The optimal growth temperature for the fungus lies between 25° and 30°C.

CONTROL Decay in harvested fruits can be held in check by storage at 10°C or below. The fungus may be seed-borne, necessitating treatment of seed before planting (1,3,4). Cultural practices are important in control. The land can be deep-ploughed so as to bury infective material, or covered with plastic sheeting to prevent contact between fruits and soil (2). Since infection is facilitated by humid conditions, it is sometimes advisable to prune dense foliage which would otherwise tend to create a moist atmosphere near the soil surface. Partial control of the disease can be achieved by the addition of certain fungi to the soil; for example species of *Trichoderma* are antagonistic to the causal fungus but otherwise harmless. A combination of various control methods is usually necessary (2). Work is in progress to breed new resistant cultivars (5).

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1 GANGOPADHYAY S. & SHARMA R.K. (1976) Spongy rot of pumpkin. *Indian Phytopathology* **29**, 423–424 illus.

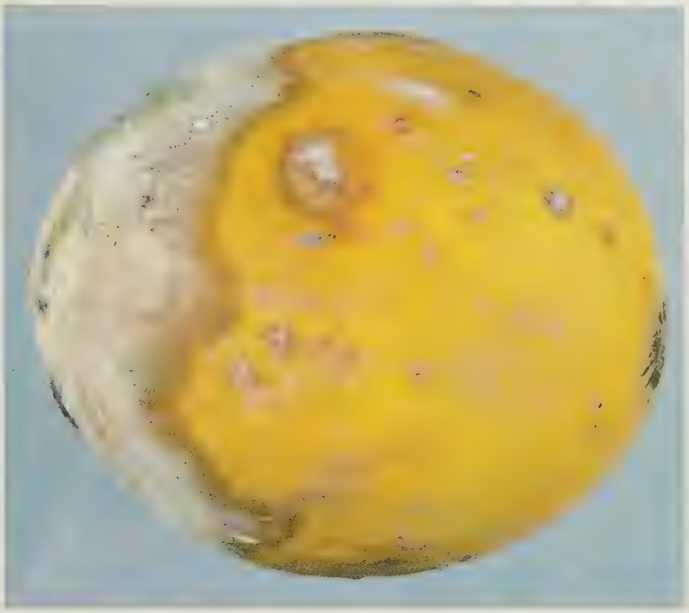
2 LEWIS J.A. & PAPAVISAS G.C. (1980) Integrated control of *Rhizoctonia* fruit rot of cucumber. *Phytopathology* **70**, 85–89 illus.

3 MADKOUR M.A., ABOU-TALEB E.M. & OKASHA A.M. (1983) Acetone inhibition of *Rhizoctonia solani* growth. *Phytopathologische Zeitschrift* **107**, 111–116.

4 MAHOLAY M.N. & SOHI H.S. (1983) *Rhizoctonia* seed rot of bittergourd and muskmelon. *Indian Journal of Mycology and Plant Pathology* **13**, 1–3.

5 SLOANE J.R., WEHNER T.C. & JENKINS S.F. (1985) Inheritance of resistance to *Rhizoctonia* fruit rot in cucumber. *HortScience* **20**, 1119–1120.

SOUR ROT of cucumbers, melons, watermelons and chayotes caused by ***Geotrichum candidum*** **Link ex Pers.**



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OCCURRENCE Sour rot has been recorded on cucumbers in Egypt (3), on melons in the USA (1), Mexico (2) and Australia (4), and (as 'waxy rot') on chayotes in India (5). Watermelons are also susceptible to attack by this fungus (1).

SYMPTOMS Lesions are soft and watersoaked, and later become covered with a flat, dull, white, powdery or waxy mould growth (2,5). In cucumbers, melons and watermelons internal tissue is not discoloured but undergoes a soft watery breakdown (1,3). In chayotes infected flesh becomes dark brown and may be watery or spongy (5). A sour odour may accompany the decay.

BIOLOGY The fungus is common in soil, and spores may be splashed on to plants during rain or carried there by insects (1). Infection occurs only via wounds, such as mechanical injuries or insect punctures, and susceptibility to rotting increases with fruit maturity (2). Fruit infected shortly before or during harvest may be packed and shipped in apparently good condition, only to decay in transit or during storage. The optimal growth temperature for the fungus is approximately 35°C, and it thrives in humid conditions with poor ventilation (2).

CONTROL It may be necessary to use an insecticide on the growing crop. Care should be taken to minimise injury to the fruits during harvesting and handling. Most fungicides are ineffective against *G. candidum*, although some of the more recent chemicals do give control (4). Refrigeration serves to inhibit decay, as the fungus makes little progress at temperatures below 10°C (2).

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1 BUTLER E.E. (1960) Pathogenicity and taxonomy of *Geotrichum candidum*. *Phytopathology* **50**, 665–672 illus.

2 CEPONIS M.J. (1966) Occurrence of *Geotrichum candidum* in western melons on the New York market. *Plant Disease Reporter* **50**, 222–224.

3 EL-TOBSHY Z.M., ABDEL-SATTAR M.A. & BARAKA M.A. (1979) Sour rot, a post-harvest disease of certain vegetables in the A.R.E. *Agricultural Research Review* **57**(2), 39–46.

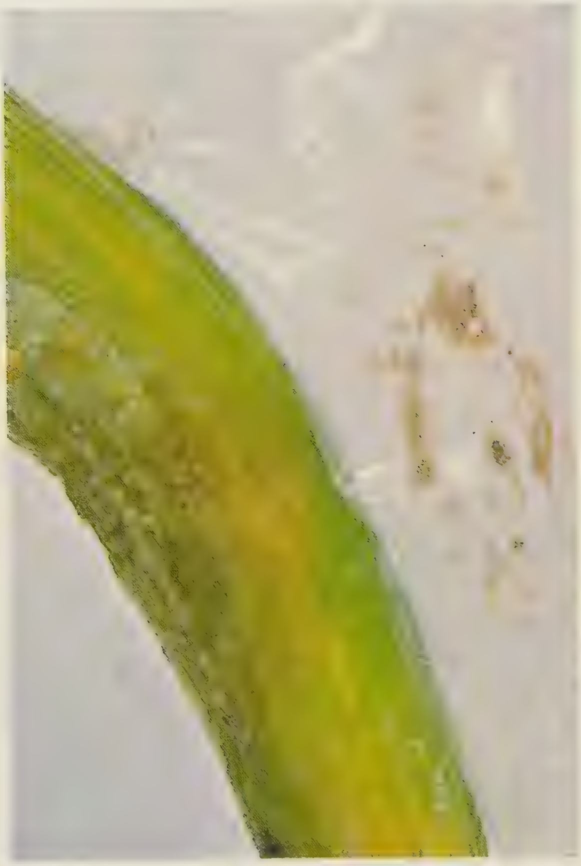
4 MORRIS S.C. & WADE N.L. (1983) Control of postharvest disease in cantaloups by treatment with guazatine and benomyl. *Plant Disease* **67**, 792–794.

5 RAO V.G. (1964) Some new market and storage diseases of fruits and vegetables in Bombay-Maharashtra. *Mycopathologia et Mycologia Applicata* **23**, 297–310.

WATERY SOFT ROT of cucumbers, watermelons, marrows, squashes and pumpkins caused by

***Sclerotinia sclerotiorum* (Lib.) de Bary**

44



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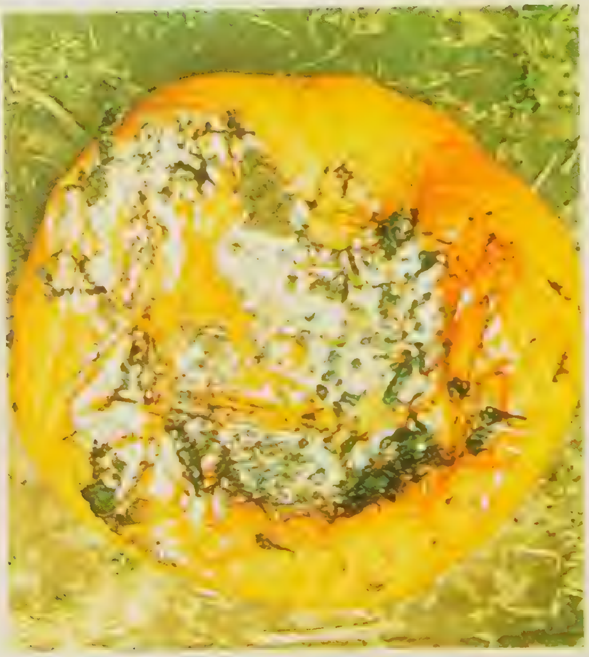
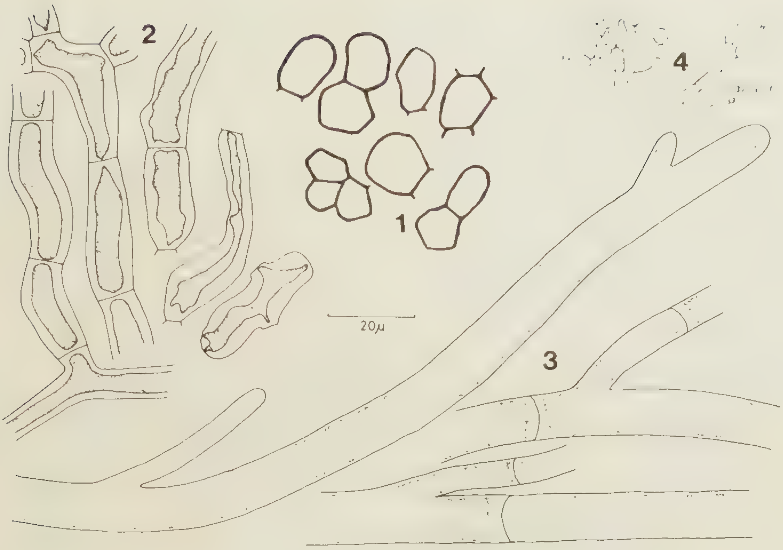


OCCURRENCE This disease is common on cucurbits grown in temperate countries or in subtropical regions possessing a cool wet season. Thus, for example, sclerotinia fruit rot has been recorded on cucumbers in the UK (5,6), Czechoslovakia (3), East Germany (1) and Japan (7); on melons in Israel (8); on watermelons in Italy (2); on squashes in Egypt (4) and Israel (9); and on squashes and pumpkins in the USA (10). *S. minor* (page 212) was presumed, but not confirmed, to be the cause of rotting in watermelons in Italy (2). All other records are of *S. sclerotiorum* and it is to this species that the following account refers.

SYMPTOMS Infected tissue is soft and becomes covered with a dense bright white mould (5). Irregular shaped bodies (sclerotia) develop; they are several mm across, white at first, exuding drops of liquid and finally turning black (4,5). The fruit tissue often undergoes a watery collapse, although in some squash cultivars a dry rot occurs, with numerous sclerotia forming in the mummified fruits (10).

BIOLOGY The sclerotia or resting bodies can survive in the soil for many years. A few weeks after the onset of rains or irrigation they 'germinate' to produce the sexual stage (apothecia giving rise to ascospores), and spores are discharged into the air (7,10). Infection occurs under moist conditions. Fruits are often infected via withered flowers which remain attached as the fruit develops to maturity (6). Harvested fruits can undergo rapid rotting in store, and the mould spreads into adjacent sound fruits, causing 'nesting'.

CONTROL If the soil is drenched with a systemic fungicide before the crop is sown, the developing plants can then absorb the chemical and thereby be protected against infection (3,8). Alternatively, or in addition, aerial sprays can be applied during the growing season (1,5,6), usually in conjunction with control measures against grey mould (q.v.). For cucurbits grown in plastic houses, the use of special film can be effective; if certain wavelengths can be cut out, 'germination' of sclerotia is inhibited and



46 *Sclerotinia sclerotiorum* (sclerotial state) (from CMI Descr. No. 513, Mordue & Holliday 1976) 1 cells from sclerotium rind 2 cells from sclerotium medulla 3 hyphae from advancing edge of colony (stained with cotton blue in lactophenol) 4 spermatia

spore production is thereby prevented (7). An alternative practice involves covering the ground with plastic sheeting so that ascospores cannot be effectively released (5). If primary control measures fail (owing, for example, to the development of fungal strains tolerant of the fungicide used) and infection is noticed in the growing crop, all infected plant parts should be promptly and carefully removed and destroyed, before sclerotia can proliferate and fall on to the soil (5). Fruits which may have incipient infection can be treated with a fungicide immediately after harvest (9) and stored under refrigeration (4).

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1 APELT G. & BOCHOW H. (1975) Zur prophylactischen Bekämpfung von *Sclerotinia sclerotiorum* (Lib.) de Bary bei Hausgurken. *Archiv für Phytopathologie und Pflanzenschutz* **11**, 101–110.
2 CIFERRI R. (1944). Marciume dei frutti d'anguria da *Pythium debaryanum. Atti del Laboratorio Crittogamico dell' Istituto Botanico, Università Pavia, Ser. 5,* **1**, 310–315.
3 DUJIČKOVÁ H. (1979) [Preparations against white rot of cucumber.] *Agrochemia, Bratislava* **17**, 228–230.
4 EL-TOBSHY Z.M. & ZAYED M.A. (1979) Post-harvest *Sclerotinia* soft rot of squash and carrot. *Agricultural Research Review* **57**(2), 47–56 illus.
5 FLETCHER J.T. (1984) Cucumber – white rot (*Sclerotinia sclerotiorum*). In *Diseases of greenhouse plants*. London: Longman, pp. 170–173 illus.
6 FRAMINGHAM E.E. & ANN D.M. (1984) Fungicidal control of *Sclerotinia sclerotiorum* on cucumber. *Tests of Agrochemicals and Cultivars* No. 5 (*Annals of Applied Biology* **104**, Supplement), 56–57.
7 HONDA Y. & YUNOKI T. (1977) Control of *Sclerotinia* diseases of greenhouse eggplant and cucumber by inhibition of development of apothecia. *Plant Disease Reporter* **61**, 1036–1040.
8 NETZER D. & DISHON I. (1970) Effect of mode of application of benomyl on control of *Sclerotinia sclerotiorum* on muskmelons. *Plant Disease Reporter* **54**, 909–912.
9 TEMKIN-GORODEISKI N. (1970) Storage rots of zucchini squash (*Cucumis pepo*) and their control. *Israel Journal of Agricultural Research* **20**, 97–99.
10 YOUNG P.A. (1936) *Sclerotinia* rot of squash and pumpkin. *Phytopathology* **26**, 184–190 illus.

OTHER DISEASES of cucumbers, melons, watermelons, marrows, squashes, pumpkins and chayotes

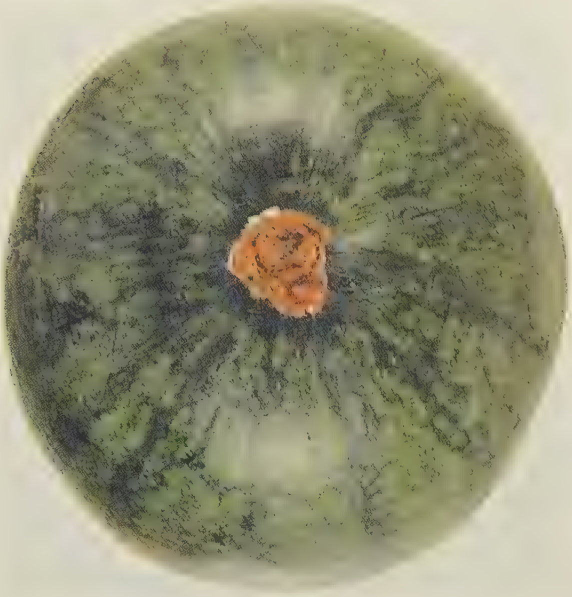
ASPERGILLUS BLACK MOULD ROT, caused by *Aspergillus niger* (page 242), has been recorded on melons from Peru (7). Lesions are soft and watersoaked, yellowish at first, and later covered with sooty black spores. The fungus is favoured by high temperatures, and can be held in check by storage at 10°C or below (10).

DOWNY MILDEW (*Pseudoperonospora cubensis* (Berk. & Curt.) Rost.) is normally confined to leaves. Occasionally it has been found on fruits, for example on greenhouse cucumbers in Italy, where it was able to cause infection only at temperatures between 17° and 22°C and relative humidities between 80 and 95% (3).

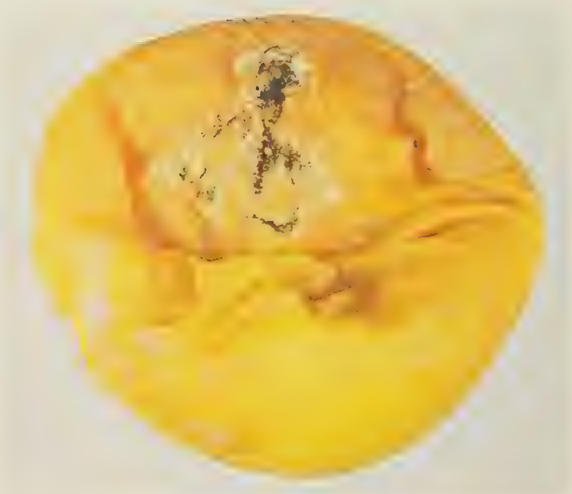
MUCOR ROT has been observed in melons removed from cold storage in the USA (10). Lesions have a dull, greasy. brownish, watersoaked appearance, and the decay may be accompanied by a sour odour. Several species of *Mucor* are capable of substantial development at 10°C.

PESTALOTIOPSIS ROT, caused by *Pestalotiopsis gubiana* Subram. & Rao, has been recorded on watermelons in India (9).

PHOMA ROT of chayote occurs in India, and *Phoma citrullicola* has been identified on Indian muskmelons (8).



47 Verticillium rot



48 Aspergillus black mould rot

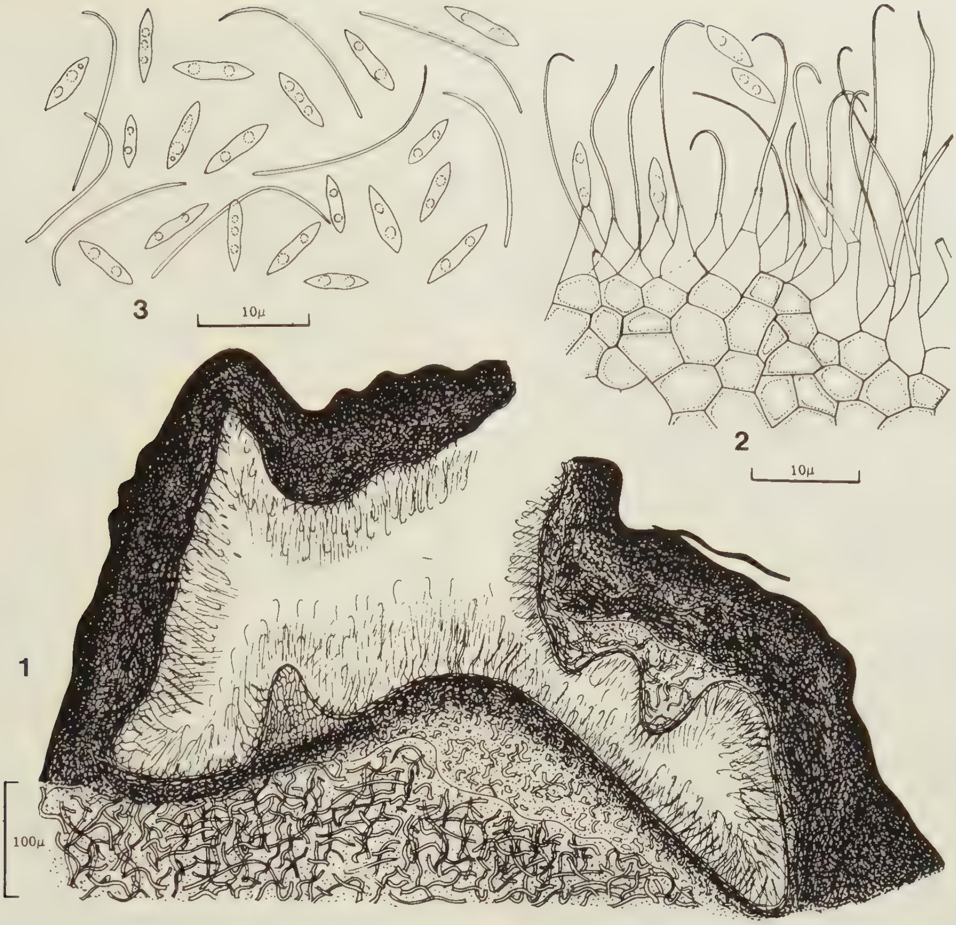
PHOMOPSIS BLACK ROT of watermelons and greenhouse cucumbers is caused by *Phomopsis cucurbitae* McKeen (5). It was closely studied in Canada after the disease had for long been confused with didymella black rot (q.v.) (5). *P. cucurbitae* has also been recorded in the UK and India, and appears to exist only in the asexual state (pycnidia giving rise to conidia). In contrast, a species of *Phomopsis* on melons in the USA occurs also in the sexual state (perithecia giving rise to ascospores) and has been named *Diaporthe melonis* Beraha & O'Brien (1). It causes a quiescent infection, and susceptibility increases with fruit maturity. Post-harvest rotting can be controlled by means of a heated fungicide dip (2).

POWDERY MILDEW of watermelons may be caused by *Sphaerotheca fuliginea* (Schlecht.) Pollacci (6). Symptoms include whitish mould spots, raised pimples, and occasionally russeted areas covering much of the fruit surface.

RED ROT of melons, caused by *Epicoccum nigrum* Link, has been recorded in the USA. The red discoloration can lead to rejection of entire consignments.

THIELAVIOPSIS ROT of melon, reported from India, is caused by *Ceratocystis paradoxa* (Dade) C. Moreau (8).

VERTICILLIUM ROT of watermelons may be caused by *Verticillium lateritium* Nees ex Fr., characterised by brick-red spores.



49 *Phomopsis cucurbitae* (from CMI Descr. No. 469, Punithalingam & Holliday 1975)
1 pycnidium 2 part of pycnidial wall, conidiogenous cells and conidia 3 conidia

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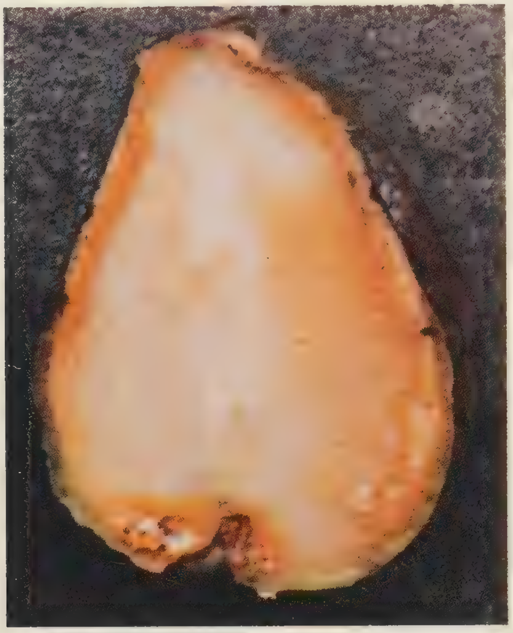
1 BERAHA L. & O'BRIEN M.J. (1979) *Diaporthe melonis* sp. nov., a new soft rot of market cantaloupes. *Phytopathologische Zeitschrift* **94**, 199–207 illus.
2 CARTER W.W. (1981) Postharvest treatment for control of stem-scar, rind and decay fungi on cantaloup. *Plant Disease* **65**, 815–816.
3 D'ERCOLE N. (1975) La peronospora del cetriolo in coltura protetta. *Informatore Fitopatologico* **25**(7), 11–13.
4 KHAN M.W. & EL-AMMARI S.S. (1987) Occurrence of three species of powdery mildews on cucurbits. *FAO Plant Protection Bulletin* **35**, 66–67.
5 McKEEN C.D. (1957) Phomopsis black rot of cucurbits. *Canadian Journal of Botany* **35**, 43–50 illus.
6 McLEAN D.M. (1970) Powdery mildew on watermelon fruits. *Plant Disease Reporter* **54**, 372–373 illus.
7 MIMBELA-LEYVA L., PASSAM H.C., REILLY P.J.A. & WALLBRIDGE A. (1975) Quality problems of South American honeydew melons imported into Britain. *Tropical Science* **17**, 61–74 illus.
8 RAO V.G. & SUBRAMONIAM V. (1975) A new fruit-rot of muskmelon. *Science and Culture* **41**, 496–497 illus.
9 SUBRAMONIAM V. & RAO V.G. (1975) *Pestalotia gubiana* sp. nov. *Norwegian Journal of Botany* **22**, 237–239.
10 WIANT J.S. (1937) Investigations of the market diseases of cantaloups and Honey Dew and Honey Ball melons. *Technical Bulletin of the United States Department of Agriculture* No. 573, 47 pp illus.

See also:

BRUTON B.D., REDLIN S.C., COLLINS J.K. & PERKINS-VEAZIE P. (1989) Red rot of muskmelon: a postharvest decay. *Phytopathology* **79**, 1196 [Abstract].

CHILLING INJURY of cucumbers, melons, watermelons, marrows, squashes and chayotes



50 Chilling injury of chayote

OCCURRENCE Critical temperatures, below which chilling injury may occur, have been reported to be between 10° and 14°C for cucumbers, watermelons, marrows, squashes and chayotes. Melons are less susceptible, and their cultivars differ substantially. Post-harvest treatments can have a significant effect on chill sensitivity (1,2,5). Work has been carried out in several countries, including Poland (1), the USA (2,5), Canada (4) and Australia (3).

SYMPTOMS Early symptoms in cucumbers and squashes may include numerous watersoaked or sunken spots (1). Melons and watermelons can develop reddish-brown patches on the skin (2,5); ripening may be prevented and taste and texture impaired. In chayotes the external sign may be a diffuse brown discoloration in the grooves of the fruit surface or, alternatively, discrete brown lesions which become pitted (3). Brown lesions may also be present about 1 cm beneath the skin, showing up in cross-section as a discoloured ring (3). In all these commodities chill-injured tissue shows an increased tendency to decay, manifest as multiple lesions affecting much of the surface area. Typical post-chilling infections are alternaria rot (q.v.) in squashes and chayotes, blue mould rot (q.v.) in cucumbers, and cladosporium rot (q.v.) in Honey Dew melons.

BIOLOGY All the cucurbits have tropical ancestors, and suffer physiological disruption at low temperature. Chilling injury can result from low temperature in the field (4), but is more usually a consequence of inappropriate storage or transit conditions (1). The time factor is critical, and a brief exposure (2 or 3 days) may cause no harm. The longer the period of exposure and the lower the temperature, the greater the damage sustained. Symptoms (which vary with the conditions imposed) (3) may not develop until the produce is returned to a higher temperature, when rapid deterioration ensues (4).

CONTROL It is not always convenient to maintain temperatures above the critical value, since at higher temperatures post-harvest life may be very short. Sensitivity to chilling injury can be reduced by plastic film wraps (cucumbers, netted melons), pre-shipment treatment with ethylene ('winter melons'), or pre-shipment 'conditioning' for a few days at moderate temperature (watermelons). Such treatments permit storage of cucumbers at 8° to 10°C, netted melons at 4°C, Honey Dew melons at 5°C, and watermelons at 7°C (5). Zucchini-type summer squashes are best kept at 8° to 10°C, while other kinds benefit from a storage temperature nearer 15°C to ensure good flavour and texture (4). Chayotes keep well at 10° to 12°C (3).

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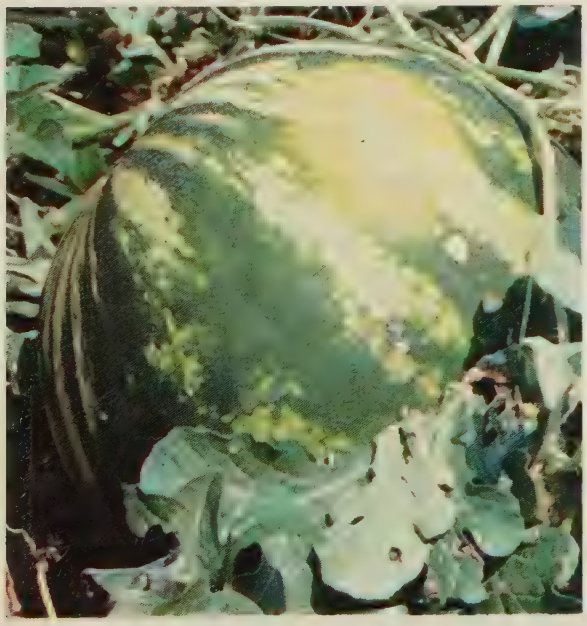
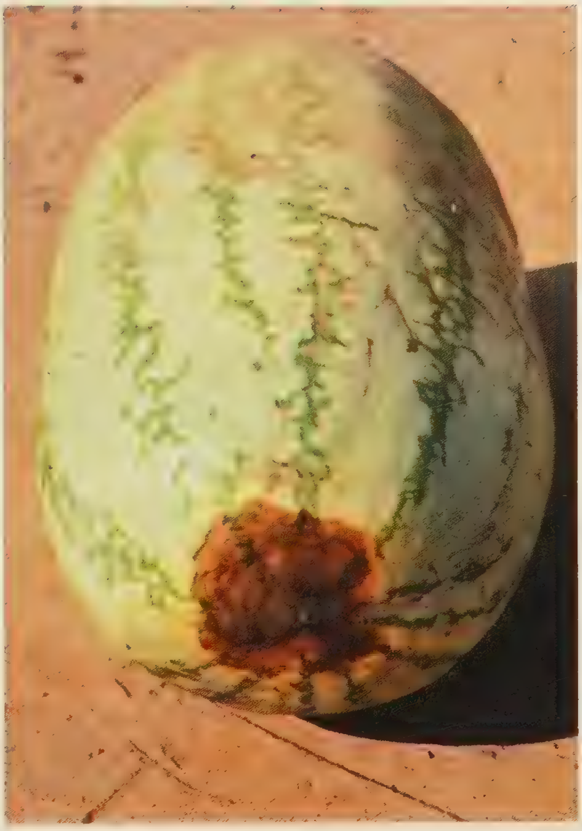
OTHER DISORDERS of cucumbers, melons and watermelons

BLOSSOM-END ROT of watermelons has recently been studied in Iraq and in Italy (1). Cylindrical-fruited cultivars are especially susceptible, the main symptom being a dark sunken area at the distal end of the fruit. The disorder develops as the fruits mature during late summer, a period characterised by high temperature, low humidity and warm dry winds. By harvest time 75% of the crop may be affected and although the disfigurement does not progress after harvest it constitutes a serious market blemish. Blossom-end rot is a nutritional disorder related to the concentration of calcium in fruit tissues; lime-deficient soils may require amendment with gypsum (calcium sulphate), and the frequency of irrigation may need to be increased to permit efficient uptake of minerals. In contrast to blossom-end rot of tomatoes (q.v.), the watermelon disorder is not aggravated by high levels of nitrogenous fertiliser. While appropriate crop husbandry can give partial control, the simplest measure is to grow spherical-fruited cultivars which are generally not susceptible to the disorder (1).

PREMATURE SENESCENCE of watermelons occurs in the presence of ethylene. Watermelons are usually harvested when already ripe (or very nearly so), and do not have much capacity for improvement in flesh quality. On the contrary, once the fruits are harvested it is not long before there is a decline in colour, texture and flavour of the ripe flesh. Such changes (fading, softening and the development of off-flavours) are accelerated by exposure to ethylene (5). It is therefore important, during shipment and storage, to segregate watermelons from fruits such as melons, tomatoes, apples and bananas which (in comparison with watermelons) produce substantial quantities of ethylene (5).

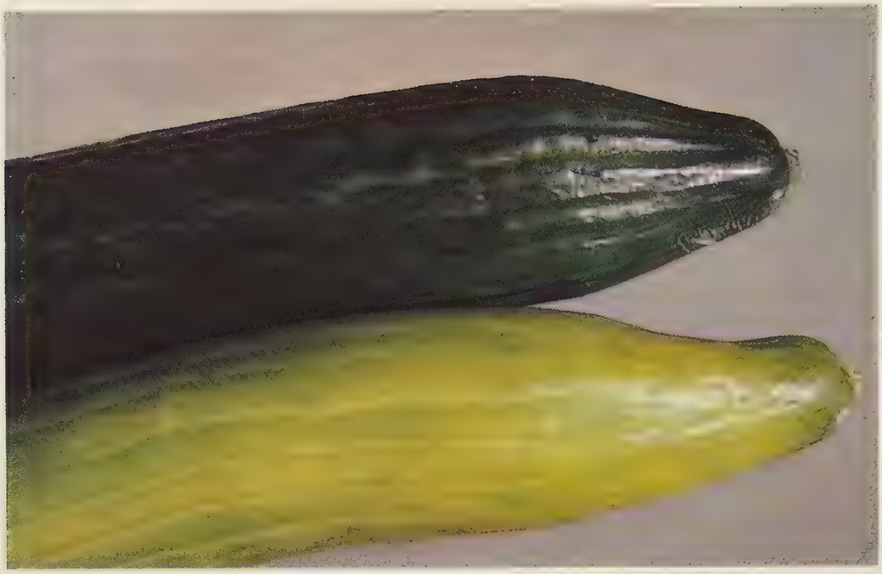


51 Solar injury of watermelon



52 Blossom-end rot of watermelon

SOLAR INJURY of melons may result from ultraviolet radiation or heat, or a combination of both (2). Symptoms on netted melons consist of a patchy ground colour or 'bronzing', and discoloration of the net. In 'winter melons', such as Honey Dew, the affected area may be brown and blotched or else greyish as if bleached. Severely injured tissue becomes sunken or wrinkled, and flesh quality may be impaired. In hot arid growing areas solar injury can be a problem in crops where there is inadequate foliage cover to shade the maturing fruits. One effective measure is to apply whitewash to the melons so that sunlight and heat are reflected (2). The whitewash is easily removed by washing the melons after harvest. Harmful exposure to the sun



53 Yellowing of cucumbers

may also occur at harvest time if fruits are not brought under cover promptly; flesh temperatures can rise several degrees above ambient air temperature, sometimes resulting in irreversible damage. It is therefore important to provide shade and to transport melons to the packhouse without delay.

VEIN-TRACT BROWNING of netted melons is a post-harvest disorder in which there is a darkening of the longitudinal grooves between the netted areas. Completely netted cultivars may suffer a similar disorder but the discoloration is less obvious. The condition is a consequence of ageing, accelerated by exposure to the sun during maturation and to high temperatures after harvest (2). Control measures are as for solar injury (q.v.). Studies in Australia have demonstrated that high humidity storage is beneficial in minimising development of this disorder.

YELLOWING of cucumbers is a sign of ageing or senescence, and is especially undesirable in fresh-market cucumbers. In 'pickling cucumbers' it is the associated flesh-softening which is deleterious, especially if accompanied by the development of off-flavours (3). These changes proceed at a rate determined by temperature, but are greatly accelerated by the presence of ethylene (4). Sound healthy cucumbers (displaying non-climacteric properties, page 12) produce little ethylene, though the rate of evolution rises in response to mechanical stress or chilling injury (3). Large fresh-market cucumbers produce less ethylene than the very immature pickling types (4). It is usually extraneous ethylene which hastens yellowing and softening in cucumbers, and the commonest source is commodities such as tomatoes, peppers and melons, which have a substantial output. If segregation is not feasible, then it is advisable to use an ethylene absorbent during shipment and storage (4).

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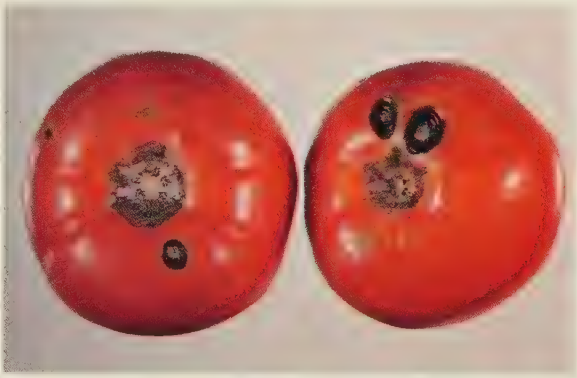
CHAPTER 2

SOLANACEOUS FRUIT VEGETABLES

The family Solanaceae provides not only one of our most important tuber crops (the potato, q.v.) but also several valuable fruit commodities. The 'exotic fruit', *Solanum muricatum*, was mentioned in Volume 1; of much greater significance, however, are tomatoes, peppers and eggplants, which are used as vegetables.

The **tomato**, *Lycopersicon esculentum* Mill. (1), originated in Peru as a small-fruited 'cherry type', var. *cerasiforme*. Subsequently the Aztecs of central America produced and maintained a diverse range of large-fruited types, and in the 16th century some of these were brought to Europe by Spanish voyagers returning from their explorations of the New World. For a long time the tomato was believed by many to be poisonous, and it was not until the 18th and 19th centuries that it began to be widely appreciated. Total world production is now some 60 million tonnes, making it one of the most important vegetable crops. The leading producer of tomatoes is the USA, followed by the USSR, China, Italy, Turkey, Egypt, Spain, Romania, Brazil and Greece. The biggest exporter of fresh tomatoes is the Netherlands, and there are also substantial exports from the Canary Islands to the UK and from Mexico to the USA. In recent years there has been a resurgence of interest in 'cherry tomatoes'; there are also plum-shaped and pear-shaped cultivars but the great majority of commercially grown cultivars are 'round' or irregularly ribbed (1). In some countries, for example Italy and the USA, a major part of the crop is grown for processing into canned tomatoes, soup, paste, sauces and other products (1).

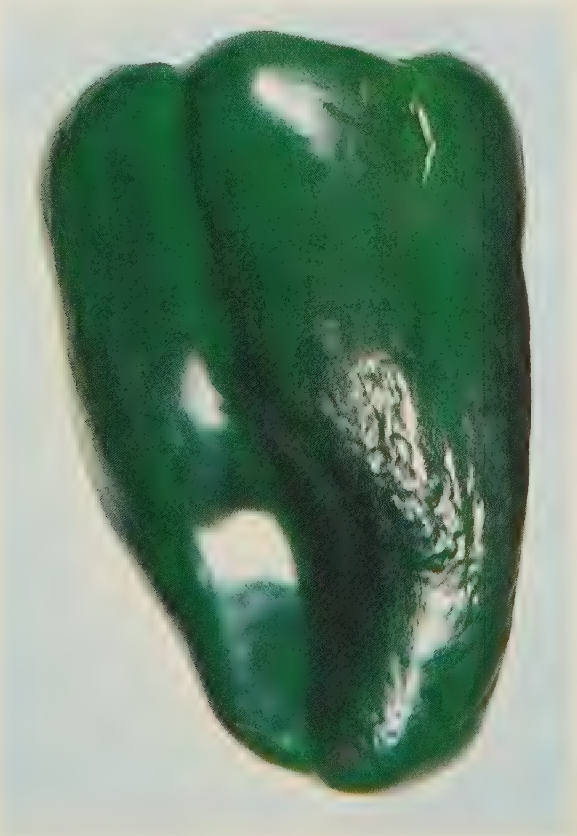
Tomatoes need warm growing conditions and in temperate countries are usually cultivated under plastic or glass, often with artificial heating; there have been sophisticated developments in soil-less culture (hydroponics) and computer-controlled environments for tomatoes grown for the fresh market (1). Outdoor crops intended for processing are left on the plant until the red-ripe stage (often artificially induced) and may then be harvested at one pass by machine. Such techniques have been made possible by the development of cultivars having a short fruit-set period and also firm fruit which can withstand the rigours of mechanical harvesting. In certain areas of the world, such as Africa and South America, these firm-fruited processing cultivars have become important in the fresh-market trade because of the excellent keeping quality of the ripe fruit (1). In contrast to processing types, typical fresh-market cultivars have an extended cropping season and serial harvesting is the norm. For distant markets fruit can be picked at the mature green stage or, for nearer outlets, at the 'breaker', 'turning', 'pink' or 'light red' stages. The tomato shows a typically climacteric pattern of respiration, and ripening of mature green fruit can be initiated by a post-harvest treatment with ethylene (6). This is a useful technique for ensuring that all fruits in a batch ripen simultaneously; however, the flavour and general quality of room-ripened but physiologically immature tomatoes is inferior to that of 'vine-ripened' tomatoes.



54 Alternaria rot of tomatoes

Being of tropical origin, the tomato is susceptible to chilling injury (q.v.) if held at too low a temperature (6). Susceptibility is influenced by stage of ripeness. Ripe fruits can safely be held for a few days at 7° to 10°C (they may be kept at lower temperatures for a longer time provided that they can be consumed immediately after storage). Light red fruits should be kept at 10°C. Mature green fruits are especially susceptible to low temperature injury and are usually held at 13°C and above. At such temperatures storage life is brief, perhaps less than 2 weeks. This period can be extended to 5 to 8 weeks by the use of a controlled atmosphere, but the application of CA storage to tomatoes has practical difficulties (4). Modified atmosphere packaging, however, (using a specific plastic film wrap) is more feasible and is effective in retarding ripening during cool-chain handling and subsequent marketing (4). For some markets the harvested fruit is left intact with calyx and short stalk in place. This is necessary in those cultivars where the calyx adheres strongly and to remove it might leave a vulnerable wound (1). The condition of calyces is a useful indication of the freshness (or otherwise) of a batch of fruit. On the other hand, as the stalks dry out they become hard and may pierce adjacent fruits with consequent injury and decay. In some markets, therefore, the preference is for fruit with calyces removed. Since there are no natural openings (stomata) in the skin of tomato fruit, gaseous exchange takes place exclusively through the calyx-stalk.

In temperate countries the major cause of post-harvest decay is grey mould rot (q.v.). In hotter growing areas the common types of decay are sour rot, rhizopus rot and bacterial soft rot (q.v.) (9). Devastating losses can occur if fruits become infected through absorption of contaminated water in dump tanks at the packhouse (2). The risk of disease is reduced but not eliminated by chlorination of the water. Additional care is necessary to ensure that the water is slightly warmer than the fruit, that depth of immersion does not exceed 60 cm, and that the fruit is submerged for no longer than 2 minutes (2). It is also important to devise means of minimising mechanical damage during handling, packing, transport and marketing (10). Research on post-harvest problems of tomatoes is receiving attention in many countries, including the USA (2,6), Israel (1), the UK (4), the Netherlands, Australia, China and Nigeria (10).



55 Chilling injury of sweet pepper

The **tree tomato** is an alternative name for the tamarillo, *Cyphomandra betacea* (Cav.) Sendtner, also within the family Solanaceae. It is considered briefly in Volume 1 (under 'Miscellaneous tropical and subtropical fruits').

The **pepper** exists in several forms. Sweet or bell peppers are the large hollow fruits of mild cultivars of *Capsicum annuum* L., and may be green, red, yellow or purple. The species also includes some cultivars of hot peppers or chillies, with varying degrees of pungency. *Capsicum frutescens* L. is a perennial which produces small fruits (usually red) which are extremely pungent. Peppers were widely spread throughout the New World tropics in pre-Columbian times, and were subsequently brought to Europe. Portuguese explorers introduced them to Asia as an interesting substitute for black pepper (the dried fruit of the vine *Piper nigrum*). Ground dried chilli is an essential constituent of curry powder. World production of sweet peppers and chillies is of the order of 9 million tonnes, and the leading producers include China, Nigeria, Turkey, Spain, Mexico, Romania, Italy, Yugoslavia, Indonesia and the USA. Figures for India are unavailable but it is an important crop there.

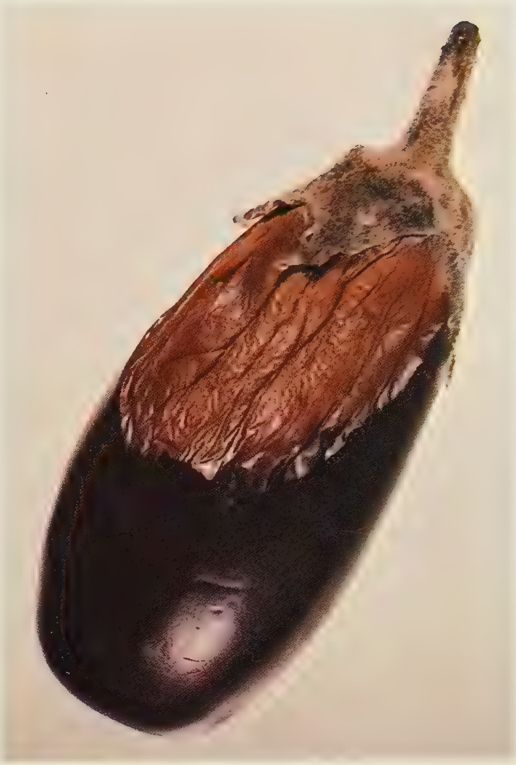
Sweet peppers are commonly harvested green. The more mature, coloured peppers are slightly less firm. In contrast to chilli peppers (5), sweet peppers show a non-climacteric pattern of respiration (neither type produces much ethylene). In order to delay colour change (from green to red) and general senescence, sweet peppers should be segregated from ethylene-producing commodities such as tomatoes and melons. The other main hazards are moisture loss (leading to wilting and premature senescence) (8) and decay (3,9). Rapid reduction of temperature helps to delay the onset of deterioration, and this may be achieved by forced air cooling, by vacuum cooling or by hydrocooling. In the last, proper chlorination of the water bath is essential for the control of bacterial soft rot (q.v.), one of the main causes of decay in peppers (3). Wax coatings and plastic film packaging help to minimise evaporative loss and may also incorporate a fungicide.

Optimal storage conditions are a high humidity and a moderate temperature so as to avoid chilling injury (q.v.). At 7° to 10°C sound healthy peppers can be held for 1, 2 or 3 weeks, depending on cultivar, state of maturity when picked, and post-harvest treatments used. A controlled atmosphere, coupled with a slightly lower holding temperature, can be used to prolong the storage life of chilli peppers awaiting processing. The more usual method of preserving chillies is to dry them to a moisture content which is sufficiently low to preclude the growth of moulds.

This is especially important because several of the fungal species which attack improperly dried material are capable of producing toxins, an example being aflatoxin from *Aspergillus flavus*.

The **eggplant** or **aubergine**, *Solanum melongena* L., is native to India, where it is called brinjal (7). It has been grown in Asia since ancient times, but was unknown to the Greeks and Romans. The Arabs brought the eggplant to Spain, several centuries after Persian travellers had introduced it into Africa. It is now grown throughout the tropics and also in cooler latitudes (often under glass or plastic). World production is over 5 million tonnes, and the main producers include China, Turkey, Japan, Egypt, Italy, Syria, Indonesia, Iraq, Spain and the Philippines. Figures for India are unavailable.

Early forms of the plant had small, pale, egg-like fruits, hence the name. Most modern cultivars, however, have large, deep purple fruits which can be oval or elongate. At the stem-end is a substantial pale green, spiny calyx. Eggplants are harvested immature and have a rather short storage life, typically 1 to 2 weeks. The optimal storage temperature lies between 8° and 12°C, the appropriate value being dependent on susceptibility to chilling injury (q.v.) of the particular crop. Waxing and high humidity storage help to minimise moisture loss which would otherwise lead to dullness and wrinkling of the skin. Exposure to ethylene is detrimental since it hastens senescence, characterised by browning, loosening and eventual abscission of the calyx. Eggplants themselves produce very little ethylene, but they can be adversely affected by ethylene evolved from other commodities such as tomatoes. Common diseases of eggplants include grey mould rot, rhizopus rot and phomopsis rot (q.v.) (7,9).



56 Rhizopus rot of eggplant

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See also:

POHRONOZNY K., WADDILL V.H., SCHUSTER D.J. & SONODA R.M. (1986) Integrated pest management for Florida tomatoes. *Plant Disease* **70**, 96–102 illus.

ALTERNARIA ROT of tomatoes, peppers and eggplants caused by ***Alternaria alternata*** **(Fr.) Keissler**

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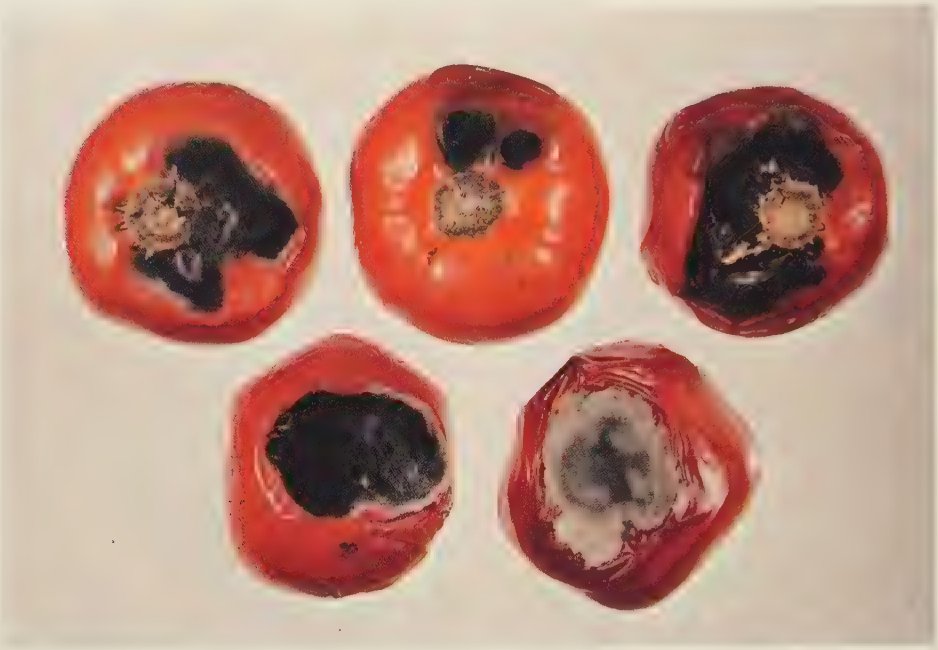

OCCURRENCE This fungus has been recorded in many countries, for example on tomatoes in France (1) and the USA (5, 9, 10), on eggplants in India (4, 6), and on peppers in Iraq (7), Israel (3) and the USA (2, 8). Other species of *Alternaria* on tomatoes include *A. solani* causing early blight rot (q.v.) and *A. syringae* pv. *tomato* causing nailhead spot (q.v.).

SYMPTOMS In tomatoes lesions caused by *A. alternata* are frequently found near the stem-scar or alternatively at the blossom-end of the fruit (1, 5). Decayed tissue is firm, dry, and brown to black, extending deep into the fruit. Internal cavities may form, lined with dark grey mould and, under humid conditions at the surface of the fruit, there may be copious production of dark olive-green to black spores (conidia). In peppers lesions tend to be at the side or at the blossom-end, and are at first watersoaked and greyish, later becoming dark, wrinkled, sunken and covered with spores. Sometimes there are no external symptoms but, if the pepper fruit is cut, dark grey mould and spores may be found growing on the seeds (2, 3). In eggplants lesions are often numerous and scattered, eventually coalescing to cover much of the fruit surface; they are dark brown at first, later becoming sunken and covered with spores (4).

BIOLOGY The fungus is very common in the air and in soil, where it survives on plant debris. It does not usually attack sound fruits which have been grown under good conditions and stored correctly after harvest. Infection can occur, however, via dying flower parts (3), insect injuries (2), cracks at the stem-end, or by way of tissue weakened through sunscald (q.v.), chilling injury (q.v.), blossom-end rot (q.v.) or overmaturity (5). Tomatoes intended for canning are left to ripen fully on the plant (1) and are particularly prone to stem-end and shoulder infections after rain or dew-formation. Fruits may undergo severe decay before they can be processed or marketed (10). The optimal growth temperature for the fungus is approximately 28°C (4) but it can make significant progress at the cold storage temperatures normally recommended for tomatoes, peppers and eggplants. A further problem is that

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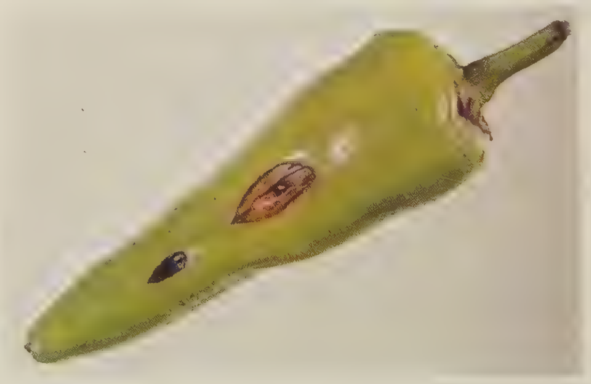

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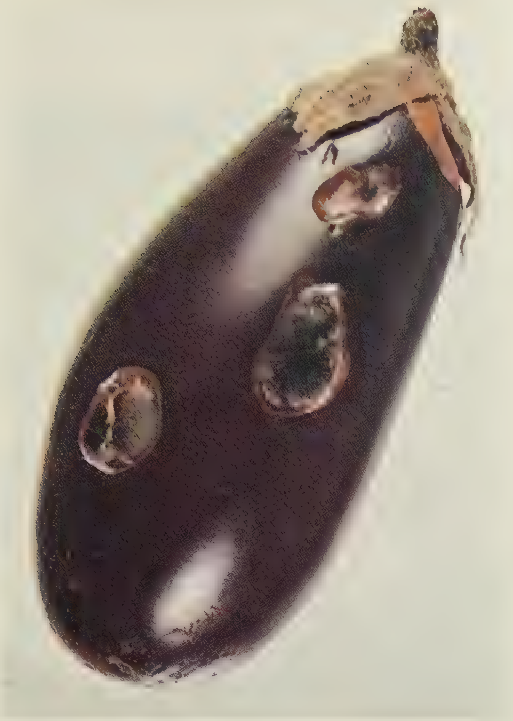

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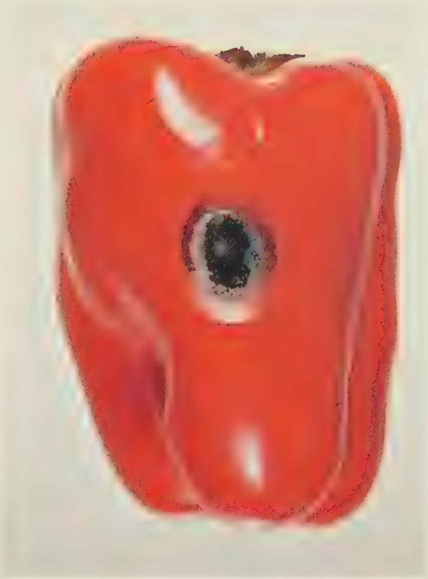

some strains of this fungus produce a toxin, necessitating especially careful quality control of fruits destined for processing (9).

CONTROL The fungus can be transmitted in seed (3,4,7) and it may therefore be advisable to treat the seed with a fungicide before planting. Certain cultivars show resistance to infection in the field, but whenever fruits are weakened in some way they become predisposed to alternaria rot. Spraying the crop with fungicide cannot be relied upon for control (10), and the most important measures include careful fertilisation and irrigation (to reduce the likelihood of blossom-end rot and cracking), and maintenance of appropriate storage temperatures for the harvested fruit (5). Development of alternaria rot can be inhibited by the use of a post-harvest fungicide dip (8). If the crop has been subjected to extremes of temperature in the field (6) (resulting in sunscald or, alternatively, chilling injury) it should be marketed without delay. The same precaution may be necessary after a wet growing season (4).

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ANTHRACNOSE of tomatoes, peppers and eggplants caused by ***Colletotrichum*** **spp.**

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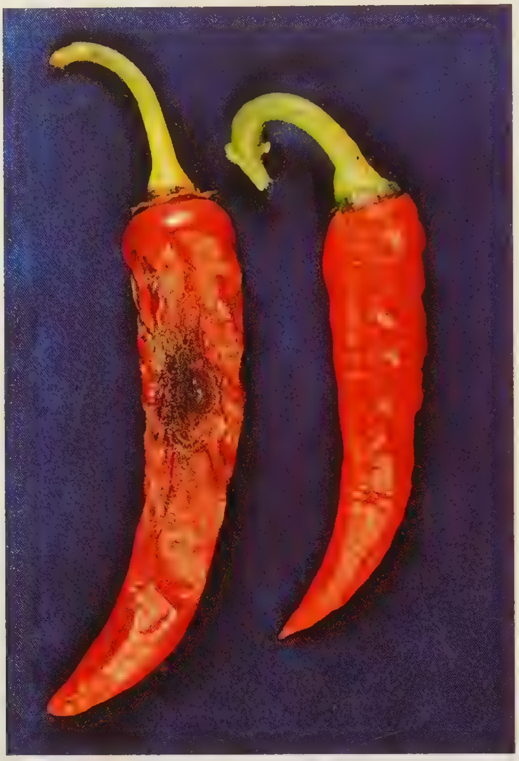

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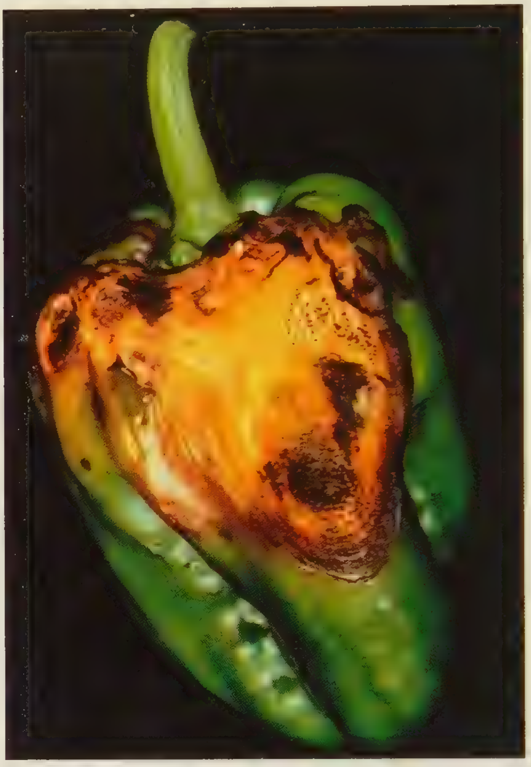
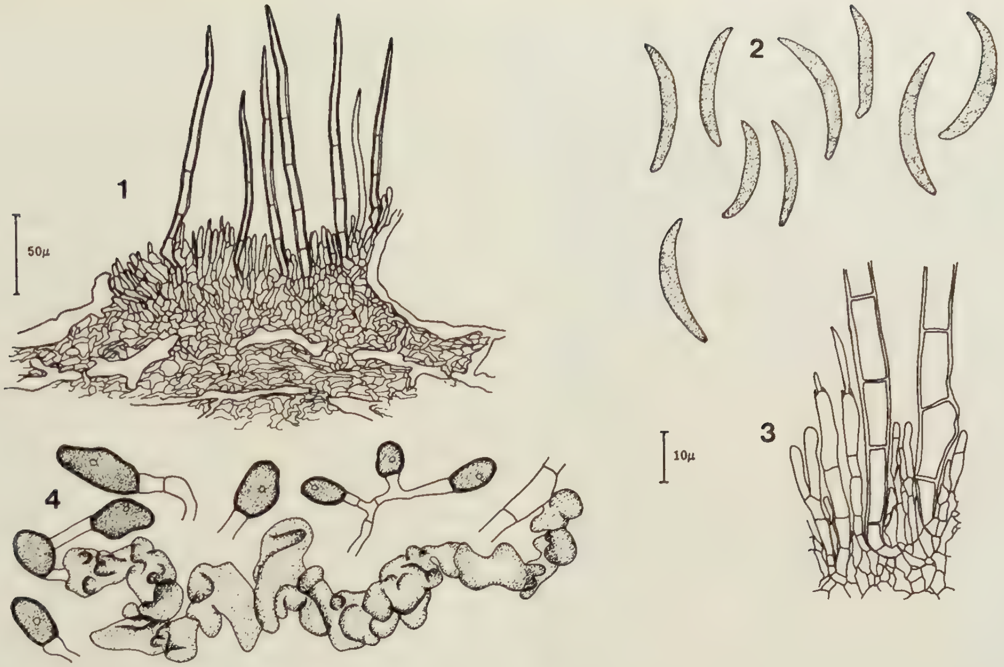


OCCURRENCE Anthracnose is an important disease which may be caused by any of several species of *Colletotrichum*. Examples are *C. capsici* (Sydow) Butler & Bisby on chilli peppers in India (5) and the southern USA; *C. coccodes* (Wallr.) Hughes (page 305) on tomatoes in Canada (3), peppers in India (10) and eggplants in Italy (8); *C. dematium* (Pers.:Fr.) Grove on tomatoes in the USA (4) and peppers in India (10); *Glomerella cingulata* (page 19) on tomatoes in Poland and (in its conidial state *C. gloeosporioides*) on tomatoes in the USA (2) and Canada (3) and on peppers in Korea.

SYMPTOMS Symptoms usually develop only on ripe fruits, hence the alternative name of 'ripe rot' (4). Lesions are at first circular watersoaked spots, later becoming dark, sunken and partially covered with salmon-pink spore masses (conidia) often in concentric circles (3). *C. coccodes* forms small black solid bodies (sclerotia) beneath the skin in the centre of the lesion.

BIOLOGY Some anthracnose fungi produce the sexual stage (perithecia giving rise to ascospores), while others exist solely in the asexual state (acervuli giving rise to conidia) or sometimes in the resting state (sclerotia). The fungi survive on weeds or on plant debris in the soil (2,3). Infection usually occurs during warm wet weather, when spores are splashed on to developing fruits. The anthracnose fungi are capable of establishing themselves on or in the skin of sound immature fruits (1), remaining in a quiescent state invisible to the naked eye. Further fungal development generally occurs only when the fruit ripens. Thus anthracnose can cause heavy losses in tomatoes and peppers grown for processing which are left to ripen fully on the plant (2). Although fruits harvested at the mature-green stage are generally resistant to anthracnose decay, the resumption of fungal growth may be triggered by storage at low temperature (3). On removal of the fruits to higher temperatures deterioration is rapid, the optimal growth temperatures for these fungi lying between 25° and 30°C. Fruit rotting results in contamination or invasion of the seeds, and the fungi remain viable even after drying and storage of the seed (9).

CONTROL It is important to remove infected crop debris promptly, and it may be necessary to apply fungicide to the seed, the soil and the growing crop (5). Post-harvest treatment helps to prolong storage



63 *Colletotrichum capsici* (from CMI Descr. No. 317, Mordue 1971) 1 acervulus 2 conidia 3 conidiophores 4 appressoria

life of fruits intended for the fresh market. Work is in progress to breed tomato cultivars with thick skins to resist infection (3,4). Some chilli pepper cultivars are resistant to anthracnose, whereas sweet peppers tend to be susceptible (7). Resistance has also been demonstrated in some commercial eggplant cultivars.

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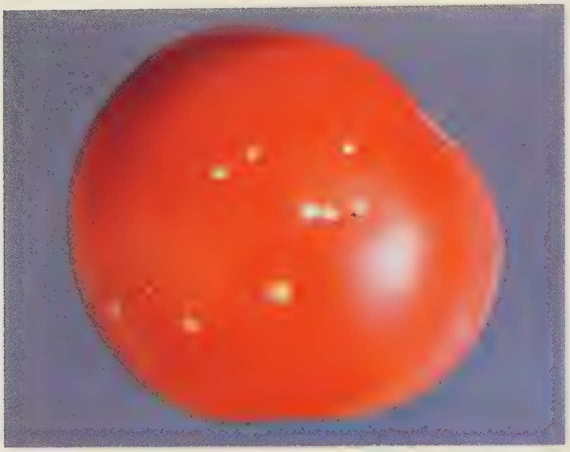
1 ADIKARAM N.K.B., BROWN A.E. & SWINBURNE T.R. (1983) Observations on infection of *Capsicum annuum* fruit by *Glomerella cingulata* and *Colletotrichum capsici*. *Transactions of the British Mycological Society* **80**, 395–401.
2 BATSON W.E. & ROY K.W. (1982) Species of *Colletotrichum* and *Glomerella* pathogenic to tomato fruit. *Plant Disease* **66**, 1153–1155.
3 ILLMAN W.I., LUDWIG R.A. & FARMER J. (1959) Anthracnose of canning tomatoes in Ontario. *Canadian Journal of Botany* **37**, 1237–1246 illus.
4 MILLER A.N., NG T.J. & BARKSDALE T.H. (1984) Comparison of inheritance of resistance to tomato anthracnose caused by two *Colletotrichum* spp. *Plant Disease* **68**, 875–877.
5 MISHRA D. (1988) Fungicidal control of anthracnose and fruit-rot (*Colletotrichum capsici*) of chilli (*Capsicum annuum*). *Indian Journal of Agricultural Sciences* **58**, 147–149.
6 PANTIDOU M.E. & SCHROEDER W.T. (1955) Foliage as a source of secondary inoculum for tomato anthracnose. *Phytopathology* **45**, 338–345 illus.
7 PEARSON M.N., BULL P.B. & SPEKE H. (1984) Anthracnose of *Capsicum* in Papua New Guinea; varietal reaction and associated fungi. *Tropical Pest Management* **30**, 230–233, 333.
8 PORTA-PUGLIA A. & MONTORSI F. (1982) Osservazioni sulla micoflora di semi di melanzana. *Informatore Fitopatologico* **32**(6), 37–41.
9 SIDDIQUI M.R., MATHUR S.B. & NEERGAARD P. (1983) Longevity and pathogenicity of *Colletotrichum* spp. in seed stored at 5°C. *Seed Science and Technology* **11**, 353–361.
10 VERMA M.L. (1982) Studies on morphology of *Colletotrichum* spp. parasitic on chillies (*Capsicum frutescens* L.) in India. *Phytopathologia Mediterranea* **21**, 97–100.

See also:

DILLARD H.R. (1989) Effect of temperature, wetness duration, and inoculum density on infection and lesion development of *Colletotrichum coccodes* on tomato fruit. *Phytopathology* **79**, 1063–1066.

BACTERIAL CANKER of tomatoes and peppers caused by
***Clavibacter michiganensis* ssp. *michiganensis* (Smith) Davis et al.**

64



OCCURRENCE Bacterial canker is one of the most serious diseases of tomato plants, and occurs in most producer countries, including Germany (3), Romania (2), Greece, Canada (5), the USA (1) and South America, and parts of Africa, Asia and Australasia. In Canada greenhouse crops tend to be more severely attacked than those grown in the open (5), while in Japan the reverse has been reported. References to bacterial canker of peppers (4) are few, and the following account refers to the disease on tomatoes.

SYMPTOMS 'Bird's-eye spotting' is the most characteristic symptom, mature lesions being small raised brown scabby spots, each surrounded by a white halo (1). When numerous lesions coalesce, the affected area feels rough and the haloes are absent. Alternatively the skin of mature green tomatoes may present a mottled appearance (as a result of systemic infection) and, if infected fruits are cut, a yellow mass of bacteria can be seen in the vascular strands.

BIOLOGY The bacteria survive in weeds, in plant debris and in the soil, and are disseminated by wind, rain and irrigation water (5). Infection occurs through pruning wounds and the bacteria spread through the vascular system. Wilting results, typically occurring only on one half of a leaf, the leaf stalk remaining firm. Minute brown cankers form on stems, leaves and calyces (1). Warm wet conditions permit rapid secondary spread, and bacteria splashed on to fruits are capable of direct penetration via hair follicles (1), giving rise to superficial bird's eye spotting (3). These small lesions do not increase in size after harvest, but nevertheless constitute a disfiguring blemish. If infection occurs via the stem-end it is likely to lead to contamination of the seed (2).

CONTROL It is essential to use clean or disinfected seed (6), and to ensure that the soil has been cleared of crop debris and, if necessary, fumigated (2). Seedlings can be dipped in an antibiotic before being transplanted. Weed control and limited use of fertilisers helps to reduce the likelihood of infection, and trickle irrigation is preferable to sprinklers (5). In Japan the lower incidence of disease in protected crops is attributed to the fact that plants are not exposed to rain. Disease outbreaks may be contained by careful removal and destruction of infected plants and those near them, together with scrupulous attention to hygiene of workers' hands, implements and clothing. In the UK, where the disease is not endemic, this bacterium is a notifiable disease organism, and seed import restrictions coupled with prompt eradication measures have prevented its establishment in this country. There are breeding programmes throughout the world, designed to produce cultivars with greater resistance to this destructive organism (4).

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2 DOCEA E. & MARINESCU G. (1977) Contribution to the control of the bacterial canker of tomato caused by *Corynebacterium michiganense* (E.F. Smith) Jensen. *Acta Horticulturae* No. 58, 469–474.

3 HEIN A. & CRÜGER G. (1962) *Corynebacterium michiganense* (E.F. Smith) Jensen als Erreger von Fruchtflecken, Blatt- und Fruchtstielnekrosen bei Tomaten. *Phytopathologische Zeitschrift* **44**, 351–360 illus.

4 HIBBERD A.M. (1986) Resistance in a pepper plant introduction to bacterial spot and bacterial canker. *Capsicum Newsletter* No. 5, 51–52.

5 McKEEN C.D. (1973) Occurrence, epidemiology, and control of bacterial canker of tomato in southwestern Ontario. *Canadian Plant Disease Survey* **53**, 127–130.

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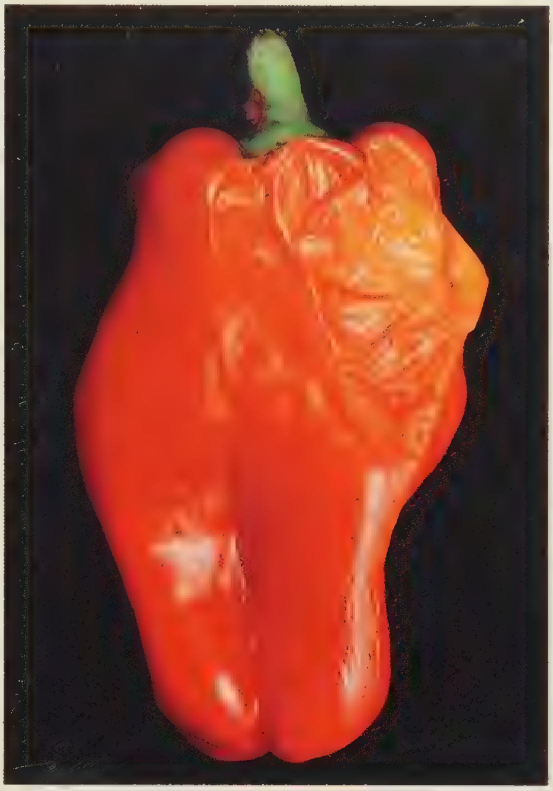
BACTERIAL SOFT ROTS of tomatoes, peppers and eggplants

OCCURRENCE Several species of bacteria cause soft rotting, which may occur in fruit from any region. *Bacillus polymyxa* has been recorded on peppers and eggplants, *Erwinia carotovora* ssp. *carotovora* on tomatoes and peppers (1,5), and *E. carotovora* ssp. *atroseptica* on peppers. Tomatoes may be infected by *Pseudomonas aeruginosa*, *P. marginalis* (3) and by *P. viridiflava*. *Cytophaga* sp. causes soft rot of bell peppers, while some strains of *Xanthomonas campestris* can cause soft rotting in tomatoes and peppers (4).

SYMPTOMS Lesions are initially soft and slightly depressed, appearing watersoaked especially near the margins. Rotting is rapid and the skin may crumple and split, with leakage of infective juices. A foul odour is often associated with this decay in tomatoes, but not in peppers and eggplants.

BIOLOGY The soft rot bacteria are to be found in soil and plant debris, and are disseminated by wind, water, insects and people. Under warm wet conditions infection of fruits occurs via wounds in the skin (6) or through the cut stem at harvest time (5). Heavy rains during the growing season increase the likelihood of infection (5). Post-harvest washing can cause infection in de-stemmed tomatoes by forcing bacteria into the stem scar through hydrostatic pressure (1). Water condensing on packaged fruit can result in increased bacterial decay (6), which then spreads from fruit to fruit. Decay is especially rapid at temperatures above 20°C.

CONTROL Treating seed with an antibiotic has been shown to reduce the incidence of bacterial soft rot of chilli peppers. Fruits should be gently handled and machinery carefully designed to minimise injuries during picking and packing. Peppers are best harvested with some stalk remaining (5). Tomatoes, on the other hand, often have the stem and calyx removed (page 54). Strict hygiene is advisable and, if fruit is washed, it is essential to change the water frequently and to ensure that it is properly chlorinated. Shallow baths are desirable, and immersion times should not exceed 2 minutes (1). Finally the fruit should be well dried before being packed, and should be kept under refrigeration during transport and storage. Controlled atmosphere storage can inhibit the growth of some species of bacteria (3).



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1 BARTZ J.A. (1982) Infiltration of tomatoes immersed at different temperatures to different depths in suspensions of *Erwinia carotovora* subsp. *carotovora*. *Plant Disease* **66**, 302–306.

2 FIORI M., CARTA C. & FRANCESCHINI A. (1982) Il "marciume molle" dei frutti di pomodoro da *Pseudomonas viridiflava* (Burkholder) Dowson. *Studi Sassaresi* III **29**, 441–447.

3 IBE S.N. & GROGAN R.G. (1983) Effect of controlled oxygen and carbon dioxide atmospheres on bacterial growth rate and soft rot of tomato fruits caused by *Pseudomonas marginalis*. *Plant Disease* **67**, 1005–1008.

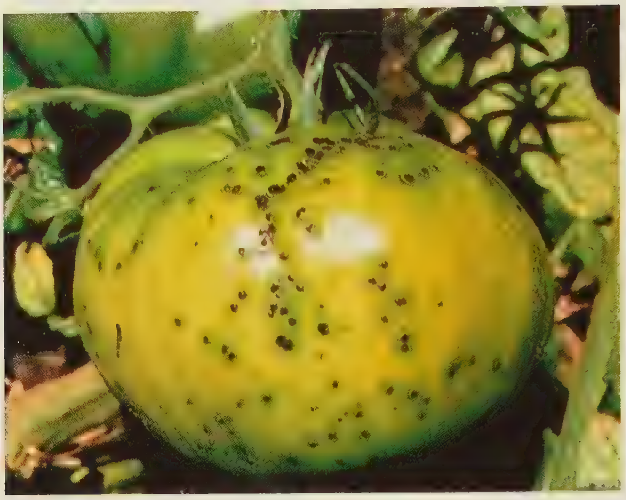
4 LIAO C.H. & WELLS J.M. (1987) Association of pectolytic strains of *Xanthomonas campestris* with soft rots of fruits and vegetables at retail markets. *Phytopathology* **77**, 418–422.

5 SHERMAN M., KASMIRE R.F., SHULER K.D. & BOTTS D.A. (1982) Effect of precooling methods and peduncle lengths on soft rot decay of bell peppers. *HortScience* **17**, 251–252.

6 VOLCANI Z. & BARKAI-GOLAN R. (1961) Soft rot of pepper fruits in storage caused by *Erwinia carotovora* var. *aroideae*. *Israel Journal of Agricultural Research* **11**, 207–208 illus.

BACTERIAL SPECK of tomatoes caused by *Pseudomonas syringae* pv. *tomato* (Okabe) Young, Dye & Wilkie

66



OCCURRENCE Bacterial speck was first described in 1933 but has assumed major importance only in recent years. It is reported from many countries, for example the USA (3,4), Canada, many parts of Europe, Israel (2), Morocco (1), South Africa, India, Australia and New Zealand (5). It may occur together with bacterial spot (q.v.) (4).

SYMPTOMS Lesions are dark, sharply delineated and typically minute (less than 1 mm). Often slightly raised, they make the skin of the fruit feel rough to the touch. Although fairly superficial, the specks constitute a serious disfigurement in fresh market tomatoes and may impair the quality of tomatoes in the processing industry. Microscopic examination reveals masses of bacteria which exude through cracks in the lesions.

BIOLOGY This bacterium persists for many years in crop debris, in the soil or on seed (2). It can also survive in weeds and in 'volunteer' tomato plants without necessarily producing symptoms (3). Cool wet weather (15° to 20°C) may precipitate disease development, and rain or overhead sprinkling aids in disseminating the bacteria which exude from leaf lesions (2). Developing fruits are infected directly, without the necessity for wounding. Mature fruits are resistant to infection and, furthermore, existing lesions are not progressive.

CONTROL Seed from an infected crop should be subjected to hot water (2) or chemical treatment (5). Field sanitation is important, and in some areas it is advisable to improve weed control. Irrigation at ground level is preferable to sprinkling (1). Chemical sprays should be applied early, and forecasting systems have been worked out to assist in correct timing. It is inadvisable to depend upon a single compound – a combination is superior. If symptoms should appear, affected plants must be promptly uprooted and destroyed. Since this disease is difficult to control, effort should be concentrated on preventive measures (3). Work is in progress to breed resistant cultivars.

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1 COLIN J., GÉRARD M. & LAABARI H. (1984) Influence du type d'irrigation sur la moucheture bactérienne chez la tomate au Maroc. *Parasitica* **40**, 3–12.

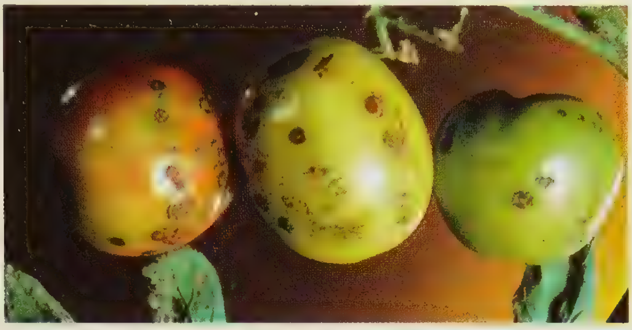
2 DEVASH Y., OKON Y. & HENIS Y. (1980) Survival of *Pseudomonas tomato* in soil and seeds. *Phytopathologische Zeitschrift* **99**, 175–185.

3 JARDINE D.J., STEPHENS C.T. & FULBRIGHT D.W. (1988) Potential sources of initial inoculum for bacterial speck in early planted tomato crops in Michigan: debris and volunteers from previous crops. *Plant Disease* **72**, 246–249.

4 McINNES T.B., GITAITIS R.D., McCARTER S.M., JAWORSKI C.A. & PHATAK S.C. (1988) Airborne dispersal of bacteria in tomato and pepper transplant fields. *Plant Disease* **72**, 575–579.

5 PYKE N.B., MILNE K.S. & NEILSON H.F. (1984) Tomato seed treatments for the control of bacterial speck. *New Zealand Journal of Experimental Agriculture* **12**, 161–164.

BACTERIAL SPOT of tomatoes and peppers caused by ***Xanthomonas campestris*** **pv.** ***vesicatoria*** **(Doidge) Dye**



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OCCURRENCE This is an important disease of tomatoes and peppers grown under warm humid conditions, for example in the southern USA (2,3,4). It has also been reported from countries in South America, Europe, Africa and Asia (CMI Descr. 20). Symptoms may be chiefly confined to the leaves, as in Israel where the disease is known as bacterial scab (1). Fruit lesions have caused direct losses of tomatoes in Florida (3) and peppers in Italy (6).

SYMPTOMS Infected tomato fruits may show dark raised spots, each surrounded by a watersoaked border. As the lesions enlarge, the margins take on a lobed appearance while the centres disintegrate and sink down, forming fibrous pits. In pepper fruits the lesions tend to remain raised and wart-like. Although fairly superficial, bacterial spot constitutes an unsightly blemish and also opens the way for secondary decay organisms.

BIOLOGY The bacteria cannot survive long in soil, but persist in solanaceous weeds (such as black nightshade) and in debris from an infected crop. Dissemination is typically by watersplash, and the bacteria invade leaves via the natural openings (stomata) (4). Fruits are infected through wounds such as insect punctures, growth cracks or abrasions, and young tissue is especially susceptible. Invasion of fruits by way of dead flower parts can result in contamination of seeds in the absence of any external fruit symptoms (1).

CONTROL Since control is difficult, efforts are directed towards prevention, often in conjunction with measures against bacterial speck (q.v.) (3). Crop rotation is helpful, provided that account is taken of the susceptibility of certain weed species. Field hygiene is essential. It is important to use clean or treated seed (1) and to ensure that transplants too are healthy. The risk of disease spreading can be minimised by the use of ground-level irrigation (in preference to sprinklers), by the application of bactericidal sprays, and by ensuring that workers do not move through the crop whilst it is still wet with dew or rain. Such measures give only partial control, however, and more subtle methods are called for. Breeding for resistance is one possibility, and some resistant tomato cultivars are available (5). Furthermore, cultivars with a sparse growth habit are more likely to escape disease than are those with dense, moisture-retentive foliage. Plants with few stomata are less likely to become infected, and even in those with many stomata, the pores can be induced to close by the application of an antitranspirant chemical (4).

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1 BASHAN Y. & OKON Y. (1986) Internal and external infections of fruits and seeds of peppers by *Xanthomonas campestris* pv. *vesicatoria. Canadian Journal of Botany* **64**, 2865–2871 illus.

2 GARDNER M.W. & KENDRICK J.B. (1923) Bacterial spot of tomato and pepper. *Phytopathology* **13**, 307–315.

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4 RAMOS L.J. & VOLIN R.B. (1987) Role of stomatal opening and frequency on infection of *Lycopersicon* spp. by *Xanthomonas campestris* pv. *vesicatoria. Phytopathology* **77**, 1311–1317.

5 SCOTT J.W. & JONES J.B. (1986) Sources of resistance to bacterial spot in tomato. *HortScience* **21**, 304–306.

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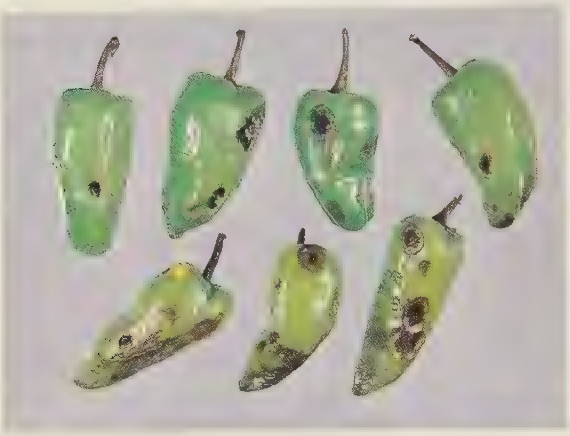
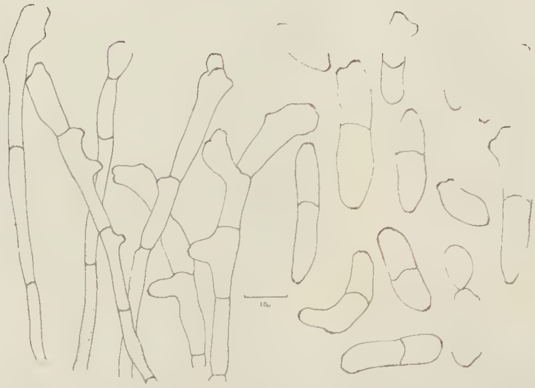
CLADOSPORIUM ROT of tomatoes, peppers and eggplants caused by

Cladosporium **spp.**
Fulvia fulva **(Cooke) Cif.**

68





69 *Fulvia fulva* (from CMI Descr. No. 487, Holliday & Mulder 1976) Conidiophores and conidia

OCCURRENCE *Cladosporium herbarum* (Pers.) Link has been recorded on tomatoes in Italy and Norway, and also on peppers in the USA (4). *C. oxysporum* Berk. & Curt. is common in the tropics and causes losses in Brazilian and Indian tomatoes (5), while *C. cladosporioides* (Fresen.) de Vries occurs on eggplants in India. *Fulvia fulva* (formerly known as *Cladosporium fulvum* Cooke) has been recorded on tomato fruits in the USA (1), the West Indies and India (CMI Map 77); this species is also notable as a cause of allergic asthma in some humans (2).

SYMPTOMS Lesions on fruits are at first circular, firm, sunken and jet black with a sharply delimited pale brown border (1,4). Under humid conditions there may be copious production of brownish-green to black spores (conidia) (4).

BIOLOGY These fungi survive in plant debris in the soil and, during humid weather, produce quantities of asexual spores (conidia) which are blown about in the atmosphere. *F. fulva* (which is important as the cause of 'leaf mould' of tomato plants) (1,2) can infect the sepals of the calyx via natural openings (stomata) and thence invade the fruit, causing a stem-end rot (1). Seeds become infected, thereby transmitting the disease to the next crop. *C. herbarum* and *C. oxysporum* are generally incapable of attacking sound tomatoes and peppers but can penetrate the skin of fruits weakened by sun scald (q.v.) or chilling injury (q.v.) (4). The latter may result from abnormally cool weather during the growing season, or from an unsuitable storage environment. These fungi can make substantial growth at low temperatures.

CONTROL *F. fulva* can be controlled by spraying the crop with fungicide and, in the greenhouse, by improving ventilation so as to reduce atmospheric humidity. Some resistant tomato cultivars are available. Infection by *C. herbarum* and *C. oxysporum* can be minimised by destroying fruits suffering from mechanical damage or sun scald, and by maintaining correct storage temperatures. If the crop has been subjected to cold weather during the growing season, especial care should be taken to avoid extended refrigerated storage (4).

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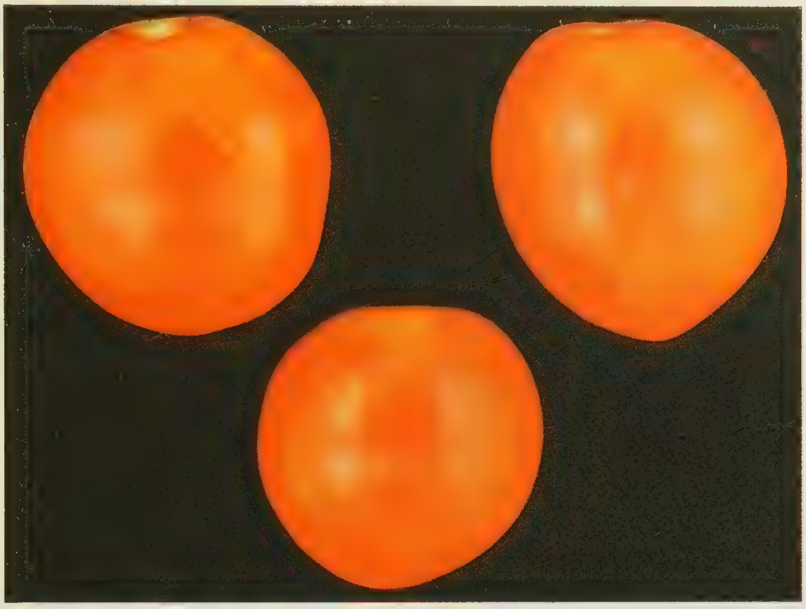
2 GUBA E.F. & RACKEMANN F.M. (1938) Species of *Cladosporium* on tomato and the allergic response in man as an aid to identification. *Mycologia* **30**, 625–634.
3 PICARD D. le, TIRILLY Y. & TRIQUE B. (1987) Antagonistes et hyperparasites du *Fulvia fulva* (Cooke) Ciferri. *Cryptogamie, Mycologie* **8**, 43–50.
4 RAMSEY G.B. & HEIBERG B.C. (1952) Cladosporium rot of peppers. *Plant Disease Reporter* **36**, 481–482 illus.
5 SHER SINGH, THAKUR D.P. & SINGH J.P. (1983) Postharvest decay of ripe tomato fruits caused by *Cladosporium oxysporum*. *Indian Phytopathology* **36**, 723–724.

COTTONY LEAK of tomatoes, peppers and eggplants caused by ***Pythium*** **spp.**



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OCCURRENCE Cottony leak occurs in warm wet growing areas. Examples are *Pythium aphanidermatum* (page 31) on tomatoes in Nigeria and in the USA (4) and on eggplants in the Philippines (2); *P. butleri* (page 329) on tomatoes in India (5); *P. debaryanum* Hesse on peppers in the USA (3); *P. myriotylum* (page 117) on eggplants in the USA (1); and *P. ultimum* (page 328) on tomatoes in the USA (4).

SYMPTOMS In tomatoes a soft watersoaked lesion may occur on any part of the fruit, enlarging rapidly until the entire fruit is infected. If the skin splits there is much leakage of juice and, since mould growth is rarely apparent, the disease is sometimes called 'water rot' (4). In peppers and eggplants decay usually begins at the blossom-end, and is often accompanied by a luxuriant growth of white cottony mould which may envelop the entire fruit. Juices leak from the rotted tissue (1,2,3).

BIOLOGY These fungi inhabit the soil, and may produce the sexual stage (oospores) as well as the asexual stage (sporangia giving rise to zoospores) (1). Infection occurs during warm wet weather when zoospores are splashed on to fruits hanging near the ground (3,4) or resting on the soil surface (2,4). Ripe tomato fruits are susceptible to direct penetration at the point of contact with infested soil (4), while peppers and eggplants are usually invaded at the blossom scar (1,3). At temperatures between 25° and 35°C rotting is rapid and, in addition to mould strands permeating infected tissue, there may also be spore-formation (1,2,3). During transport and storage infected tomatoes tend to undergo a wet collapse, while peppers and eggplants exhibit 'nesting' (2).

CONTROL The only means of control is to prevent contact between fruits and soil (4), by staking the plants or by covering the ground with a mulch of plastic sheeting.

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1 DRECHSLER C. (1943) Two species of *Pythium* occurring in southern states. *Phytopathology* **33**, 261–299 illus.

2 ILAG L.L. (1976) The cottony leak disease in the Philippines. *Plant Disease Reporter* **60**, 12–13 illus.

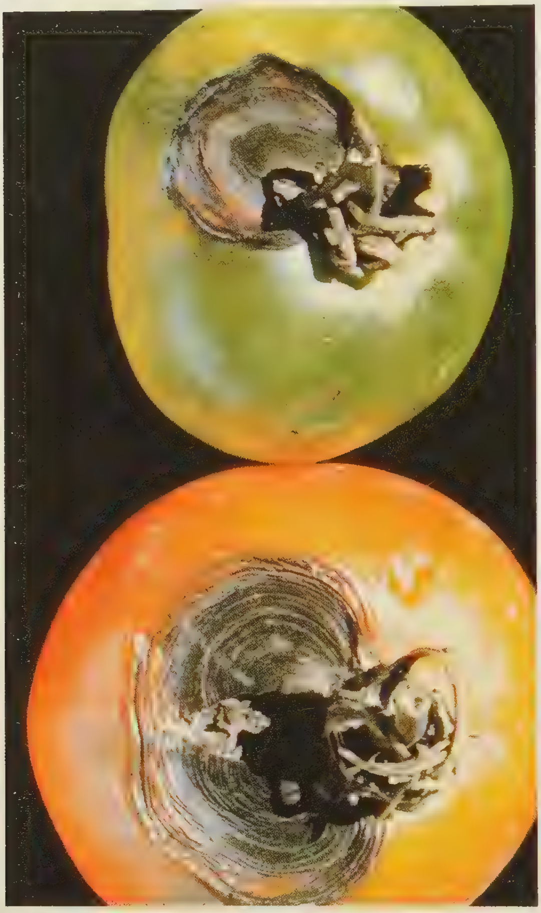
3 LEHMAN S.G. (1921) Soft rot of pepper fruits. *Phytopathology* **11**, 85–87.

4 PEARSON R.C. & HALL D.H. (1973) Ripe fruit rot of tomato caused by *Pythium ultimum* and *Pythium aphanidermatum*. *Plant Disease Reporter* **57**, 1066–1069 illus.

5 TRIPATHI N.N. & GROVE R.K. (1977) Pathogenic variability in *Pythium butleri*. *Indian Phytopathology* **30**, 222–228.

EARLY BLIGHT ROT of tomatoes caused by *Alternaria solani* Sorauer

71



OCCURRENCE Early blight has been reported from most tomato-producing countries, including Canada (1), the USA (2), Egypt (3), Germany (5) and Australia (2). The same fungus causes early blight of potatoes (q.v.).

SYMPTOMS Lesions on tomatoes are most common at the stem-end, although they may also be found at the blossom-end or on the side of the fruit (4). The affected area is dark brown; in green fruit lesions tend to be sunken and leathery with concentric markings, while in ripe fruits a soft rot occurs (4,5). There may be secondary infection by *Alternaria alternata* (page 101).

BIOLOGY *A. solani* produces thick-walled resting spores (chlamydospores) (1) which survive in crop debris and are very resistant to high temperatures and drought (5). Abundant asexual spores (conidia) are produced when short spells (1 to 3 days) of warm dry weather occur during longer moist periods. The spores are dispersed by air currents, and commonly cause leaf-spotting. In desert areas dew or overhead irrigation can provide sufficient moisture for infection, which may be particularly severe if the plants have been damaged by wind-blown sand. Any factor which reduces the vigour of the plant tends to predispose it to infection, and in temperate areas crops grown in waterlogged soils are especially vulnerable. Severe leaf infection can result in defoliation; stems may also be attacked, leading to invasion of fruits (1). Developing fruits are more susceptible than ripe fruits (3). Seed from an infected crop may be contaminated.

CONTROL It may be advisable to treat the seed and the seed-bed with a fungicide before planting. Monitoring of ambient temperature and humidity during the growing season permits forecasting of disease outbreaks, so that preventive sprays can be applied (2). At the end of the season all crop debris should be removed and destroyed. Cultivars differ significantly in susceptibility to early blight (1,3).

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1 BASU P.K. (1974) Measuring early blight, its progress and influence on fruit losses in nine tomato cultivars. *Canadian Plant Disease Survey* **54**, 45–51.

2 PENNYPACKER S.P., MADDEN L.V. & MacNAB A.A. (1983) Validation of an early blight forecasting system for tomatoes. *Plant Disease* **67**, 287–289.

3 REDA F., MOUSA O.M., SEJINY M.J. & NAWAR L.S. (1985) Response of tomato cultivars to infection with *Alternaria solani* in relation to growth and chemical composition of their fruits. *Egyptian Journal of Phytopathology* **17**(2), 83–93.

4 SAMUEL G. (1932) *Macrosporium solani* on tomato fruit. *Phytopathology* **22**, 613–614 illus.

5 TESCHNER G. (1953) Untersuchungen über *Alternaria solani*, den Hartfäule-Erreger der Kartoffel und Fruchtfäule-Erreger der Tomate. *Phytopathologische Zeitschrift* **21**, 133–162 illus.

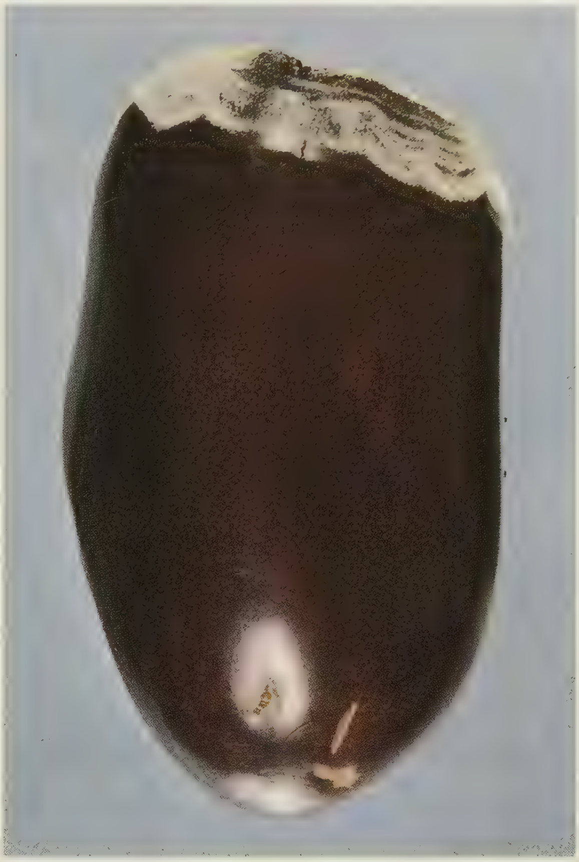
FUSARIUM ROT of tomatoes, peppers and eggplants caused by ***Fusarium*** **spp.**

OCCURRENCE Several species of *Fusarium* have been recorded, for example *F. avenaceum* on tomatoes in Italy (4); *F. equiseti* (page 35) on tomatoes in Egypt and on peppers in Nigeria (1); *F. moniliforme* on peppers in Nigeria and on eggplants in India; *F. oxysporum* on tomatoes in Canada (3), on peppers in the Philippines (5) and in Brazil, and on eggplants in India; *F. pallidoroseum* on tomatoes in the West Indies and on eggplants in India (2); *F. solani* on peppers in the Philippines (5) and Brazil and on peppers and eggplants in India; and *F. solani* var. *coeruleum* on eggplants in Israel.

SYMPTOMS On tomatoes harvested at the mature green stage and ripened subsequently, the decayed area is watersoaked, slightly sunken but still firm, often with white, pinkish or yellowish mould growth on the lesion. Infected flesh is pale brown and sharply delimited from surrounding healthy tissue. In tomatoes ripened on the plant fusarium rot tends to be soft and watery, with characteristic external mould growth.

BIOLOGY These fungi are common in soil and plant debris, and their spores (conidia) are dispersed by wind and water. Infection occurs only if fruits have been weakened in some way, for example by mechanical damage during harvest. After harvest, sound fruits contaminated with spores become susceptible to infection if stored at too low a temperature. Tomatoes stored in controlled atmosphere coupled with high humidity are predisposed to fusarium rot (3).

CONTROL The fungus may be seed-borne; seed treatment may therefore be advisable. Spraying the crop with palm-kernel oil is reported to give some control of fusarium rot of peppers (1), and a post-harvest dip in such oil can protect the peppers during storage at ambient temperature in the tropics (1). Care should be taken to prevent injury to the fruit during harvesting. A post-harvest fungicide dip can be effective, followed by storage at the correct temperature (pages 53–55). It has been shown that the addition of ethylene has the effect of suppressing fusarium rot of tomatoes in controlled atmosphere storage (3). Modified atmosphere packaging (using plastic film) also has the effect of inhibiting decay.



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1 ADISA V.A. & LEKUNZE J.K. (1986) Fruit rots of *Capsicum annuum* and *C. frutescens* in Nigeria. 2. Their control by oils. *Fitopatologia Brasileira* **11**, 817–822.

2 KAUR S., KAUR R., KAUR P. & DILBAGH SINGH (1985) Studies on wilt and fruit rot of brinjal caused by *Fusarium semitectum*. *Indian Phytopathology* **38**, 736–738.

3 LOCKHART C.L. (1970) Suppression by ethylene of *Fusarium oxysporum* growth in culture and rots of tomato in controlled atmosphere storage. *Canadian Journal of Plant Science* **50**, 347–349.

4 MARRAS F., CORDA P. & FIORI M. (1979) *Fusarium roseum* var. *avenaceum* (Sacc.) Snyd. et Hans., agente di un marciume molle dei frutti di pomodoro in coltura protetta. *Studi Sassaresi III* **27**, 233–242.

5 MICOSA R.S. & ILAG L.L. (1977) Fruit rot of pepper caused by *Fusarium* spp. *Philippine Phytopathology* **13**(1/2), 14–23.

GHOST SPOT of tomatoes and **GREY MOULD ROT** of tomatoes, peppers and eggplants caused by

Botryotina fuckeliana **(de Bary) Whetzel**
Conidial state: *Botrytis cinerea* Pers.

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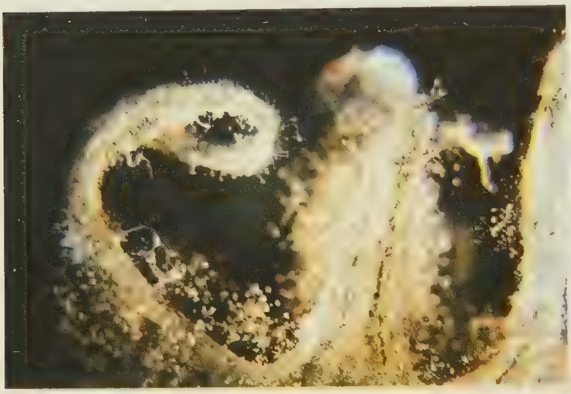

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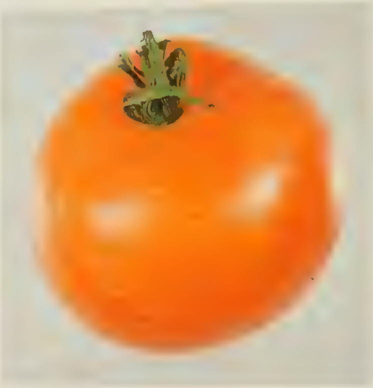

OCCURRENCE The grey mould fungus is common in temperate regions and in sub-tropical countries possessing a cool wet season. Ghost spot of tomatoes has been closely studied in the USA, the UK and the Netherlands (4,10). Grey mould rot has been reported both in outdoor crops (2) and in greenhouse crops (1) in countries such as the USA (2,7), Israel (3), the UK (5,8), the Netherlands (4,10), Italy (1), Spain (6) and New Zealand (9). Grey mould is frequently found in conjunction with sclerotinia disease or watery soft rot (q.v.).

SYMPTOMS Ghost spotting of tomatoes is characterised by pale circular haloes usually on the shoulder of the fruit (4,10). At the centre of each halo a dark point, often raised, may be apparent, and when lesions are numerous the most conspicuous feature is a roughness of the affected skin. Ghost-spotting is non-progressive in harvested fruits (4). Grey mould lesions may occur anywhere on the fruits, including the stem-end and the blossom-end (3,6); in tomatoes and peppers infected tissue is watersoaked and brownish-grey, while in eggplants the lesions are brown and sometimes have a reddish-purple margin. Under humid conditions there is copious development of grey-brown spores (conidia), sometimes accompanied by small black bodies (sclerotia).

BIOLOGY The fungus can persist in the soil as resting bodies (sclerotia) or in infected plant debris. Under cool moist conditions quantities of asexual spores (conidia) form on moribund vegetation and are disseminated by air currents. Young tomato fruits are susceptible to direct penetration by germinating spores, but further fungal growth is arrested and symptoms are restricted to disfiguring blemishes known as 'ghost spots' (4,10). If fruits of tomato, pepper or eggplant are in direct contact with the soil, infection is facilitated and decay follows (2). Alternatively the fungus may first colonise dying flowers (1,3,6) and subsequently cause rotting at the blossom-end of the fruit or any part on which cast blossoms may lodge (1). Sometimes the fungus gains entry through growth cracks, wounds, or

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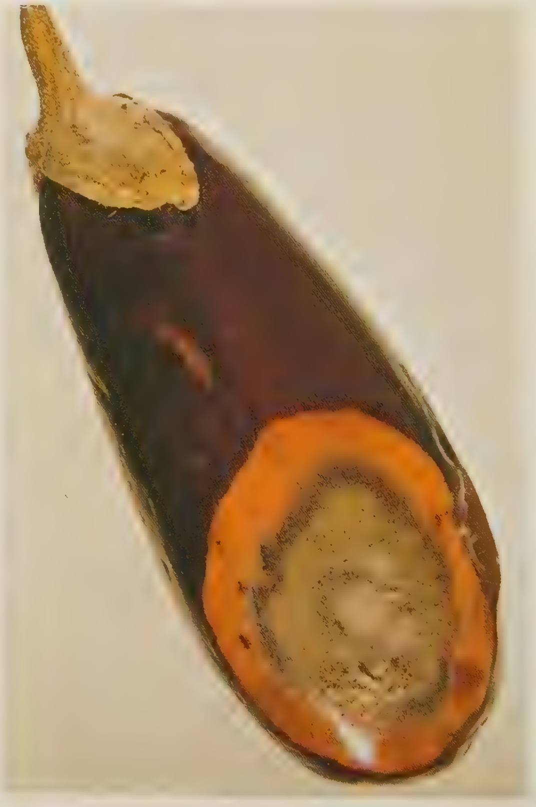

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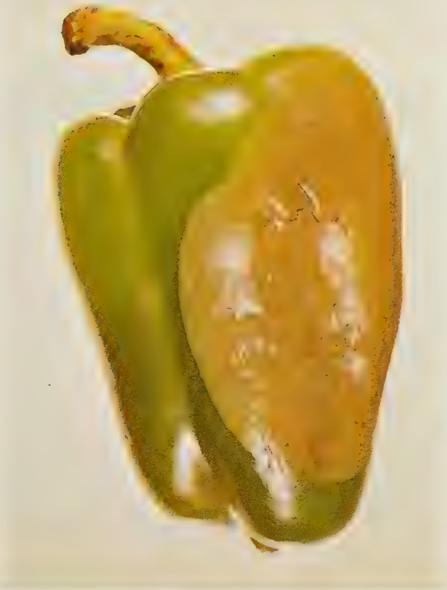

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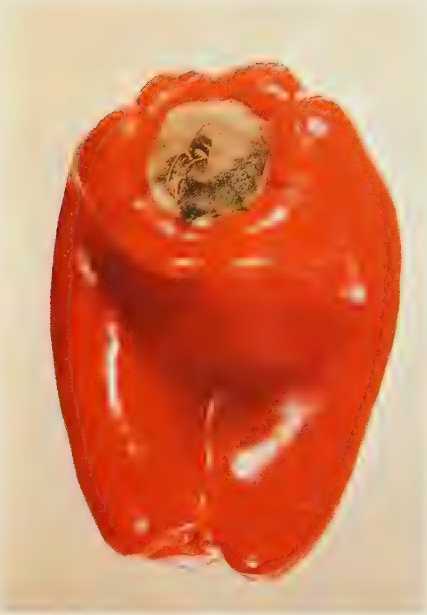

through the stem-end before or during picking. If harvested fruits are subjected to chilling injury (q.v.) the skin becomes susceptible to direct invasion (7). Subsequent removal to a warmer environment results in rapid enlargement of multiple lesions.

CONTROL It is important to remove plant debris on which the fungus could proliferate. A novel way of reducing the number of spores is to construct greenhouses from special ultra-violet-absorbing film which inhibits spore production; however, it is reported that efficacy may be short-lived owing to the development of new fungal strains (9). In protected crops efficient ventilation is essential as a means of reducing atmospheric humidity (8), and it may be feasible to install hot air fans. Fungicide sprays and (for greenhouse use) fumigants are widely used (1), and post-harvest treatments have been used successfully (2). Repeated use of a particular chemical may, however, result in the development of insensitive fungal strains (5), and it is preferable to alternate between different substances. Care should be taken to avoid chilling injury during transport and storage (7).

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2 CHASTAGNER G.A. & OGAWA J.M. (1979) A fungicide-wax treatment to suppress *Botrytis cinerea* and protect fresh market tomatoes. *Phytopathology* **69**, 59–63.

3 LAVY-MEIR G., BARKAI-GOLAN R. & KOPELIOVITCH E. (1988) Initiation at the flowering stage of postharvest *Botrytis* stem-end rot in normal and non-ripening tomato fruits. *Annals of Applied Biology* **112**, 393–396.

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6 LÓPEZ H., C.J., SAGASTA A., E.M. & GRANA E., E. (1986) Susceptibilidad de plantas de pimiento (*Capsicum annuum* L.) y berenjena (*Solanum melongena* L.) a la infección por *Botrytis cinerea*, en invernadero. *Phytopathologia Mediterranea* **25**(1–3), 135–139.

7 McCOLLOCH L.P. & WRIGHT W.R. (1966) Botrytis rot of bell peppers. *Marketing Research Report of the United States Department of Agriculture* No. 754, 9 pp illus.

8 MORGAN W.M. (1985) Influence of energy-saving night temperature regimes on *Botrytis cinerea* in an early-season glasshouse tomato crop. *Crop Protection* **4**, 99–110.

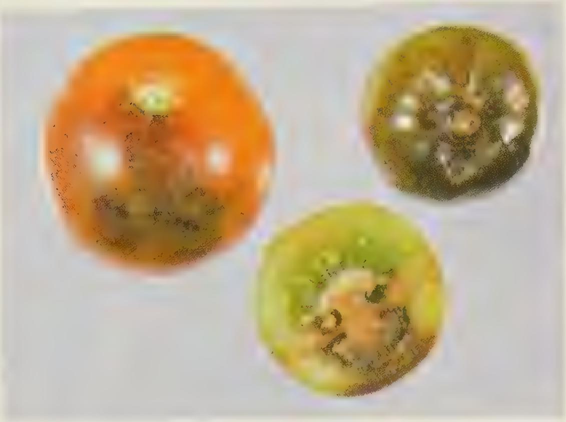
9 STEWART T.M. & LONG P.G. (1987) Sporulation of *Botrytis cinerea* in the dark. *New Zealand Journal of Experimental Agriculture* **15**, 389–394 illus.

10 VERHOEFF K. (1970) Spotting of tomato fruits caused by *Botrytis cinerea*. *Netherlands Journal of Plant Pathology* **76**, 219–226.

LATE BLIGHT ROT of tomatoes caused by

***Phytophthora infestans* (Mont.) de Bary**

78



OCCURRENCE Late blight can cause heavy losses in tomatoes grown under mild moist conditions, whether the crop is protected or in the open. Occasional epidemics of the disease have been recorded in temperate countries such as the UK, the Netherlands (4), Germany (1) and Switzerland (3), and during cool wet seasons in tropical or subtropical regions, for example India and Nigeria (2).

SYMPTOMS Lesions may occur anywhere on the fruit but are most commonly found at the stem-end. The affected area is characteristically hard, lumpy, reddish-brown and blotchy, with an indefinite margin; under humid conditions a fine white mould develops and spores may be formed. Softening occurs only if the tissue is invaded by secondary organisms. In contrast to the wet phytophthora rot (q.v.) caused by other species, late blight rot is sometimes called 'dry phytophthora rot'.

BIOLOGY *P. infestans* is also the cause of potato blight (q.v.), and tomatoes grown near to infected potato crops are therefore especially vulnerable (2). Cool wet nights and warm moist days permit the formation and dispersal of vast quantities of spores (or, more correctly, sporangia) which cause infection of leaves, stems and fruits. The optimal temperature for infection is between 15° and 18°C and, if fruit remains wet for several hours, the fungus is capable of direct penetration of the skin (1). Usually, however, invasion occurs at the edge of the stem scar. During epidemics the entire crop may rot in the field; when the disease is less severe, fruits with slight blemishes may be harvested, packed and shipped. Infected fruits decay in transit, but the fungus does not spread into surrounding healthy fruits.

CONTROL Since disease development is largely determined by the environmental factors temperature and humidity (which are influenced by rain or dew), these may be monitored as a means of forecasting potential outbreaks. The critical conditions vary slightly in different regions; the establishment of an accurate local forecasting system permits the timely application of preventive fungicide sprays. A mixture of chemicals is recommended (3). In greenhouse crops it may also be advisable to reduce the humidity by improving ventilation. After harvest, crop debris should be collected and destroyed, including diseased potato tubers from neighbouring fields. Although the fungus does not survive in dry commercial tomato seed, it can persist in the moist seeds of discarded rotten fruits, which should therefore be removed (5). Some tomato cultivars are partially resistant to the late blight fungus (4).

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1 EGGERT D. (1970) Das Verhalten von Tomatenfrüchten nach Infektion durch *Phytophthora infestans. Phytopathologische Zeitschrift* **67**, 112–128 illus.

2 ERINLE I.D. & QUINN J.G. (1980) An epiphytotic of late blight of tomatoes in Nigeria. *Plant Disease* **64**, 701–702.

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NAILHEAD SPOT of tomatoes caused by ***Alternaria tomato*** **(Cooke) Weber**

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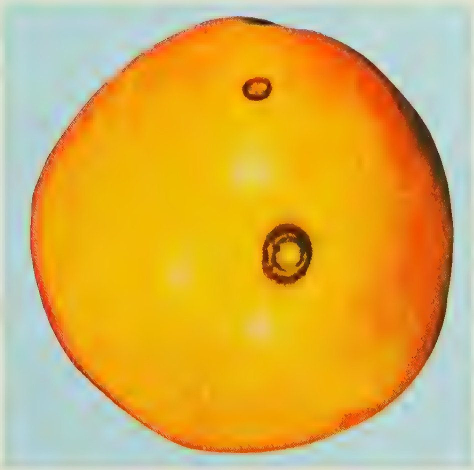

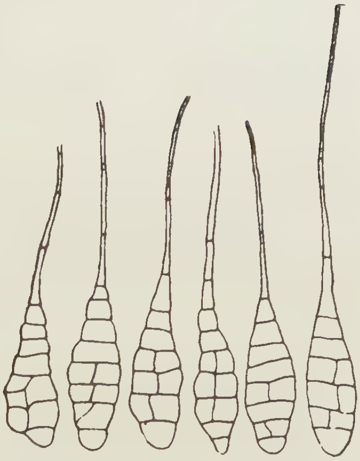

80 Conidia of *Alternaria tomato* (from *Phytopathology* **10**, 9–22, Rosenbaum 1920)

OCCURRENCE Nailhead spot has been recorded on tomatoes grown in the southern USA, Mexico, Cuba, the West Indies, Czechoslovakia, Denmark and Egypt (5). The disease was very destructive in the 1920s but virtually disappeared after the introduction of resistant tomato cultivars (2). However, occasional outbreaks have occurred in recent years (1).

SYMPTOMS Shallow greyish-brown spots may appear anywhere on the skin, and gradually enlarge until they are a few mm in diameter (5). Mature lesions are sunken and fissured, with a narrow black border and a paler centre; the surrounding tissue may remain green, and the general appearance is one of delicate concentric rings (1).

BIOLOGY The fungus survives on plant debris, and its spores (conidia) are dispersed in air currents or by water, insects and field workers. Warm moist conditions permit infection of all aerial parts of the tomato plant (2). Fruits of any age may be directly invaded, although by the time they reach the mature green stage the skin has become less susceptible to attack (4). Infection may also occur through wounds (4). Lesions which are insignificant at the time of harvest can enlarge during shipment and storage, and new lesions may develop, especially on the smaller sized fruits (3). The optimal growth temperature for the fungus is approximately 25°C. Secondary organisms often cause rapid breakdown of the entire fruit (5).

CONTROL Complete control of this disease was achieved by the introduction of resistant cultivars, and the fungus (which appears to attack only tomatoes) consequently declined (2). However, modern cultivars may be susceptible and, after a recent outbreak of nailhead spot in the USA (1), it was surmised that the fungus probably originated in seed imported from a subtropical source. It did not survive the winter, and subsequent tomato crops were free from the disease (1).

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1 GOODE M.J. & MONTGOMERY F.W. (1979) Outbreak of nailhead disease of tomatoes in southeast Arkansas. *Arkansas Farm Research* **28**(4), 9.

2 PAULUS A.O. & POUND G.S. (1955) Effect of air temperature on initiation and development of gray leaf spot and nailhead spot of tomato. *Phytopathology* **45**, 168–174.

3 RAMSEY G.B. & BAILEY A.A. (1929) Development of nailhead spot of tomatoes during transit and marketing. *Journal of Agricultural Research* **38**, 131–146 illus.

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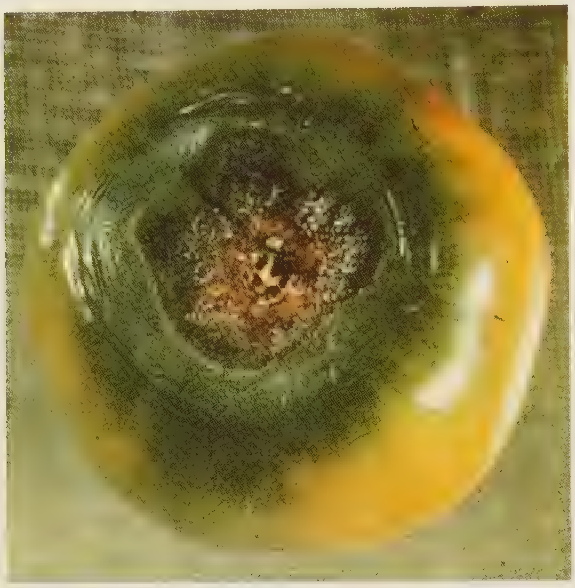
5 WEBER G.F. (1939) Nailhead spot of tomato caused by *Alternaria tomato* (Cke.) N. Comb. *Bulletin of Florida Agricultural Experiment Station* No. 332, 54 pp illus.

PHOMA ROT of tomatoes, peppers and eggplants caused by

Didymella lycopersici **Kleb.**
Conidial state: *Phoma lycopersici* Cooke
Phoma destructiva **Plowr.**
Phoma exigua **Desm.**

81

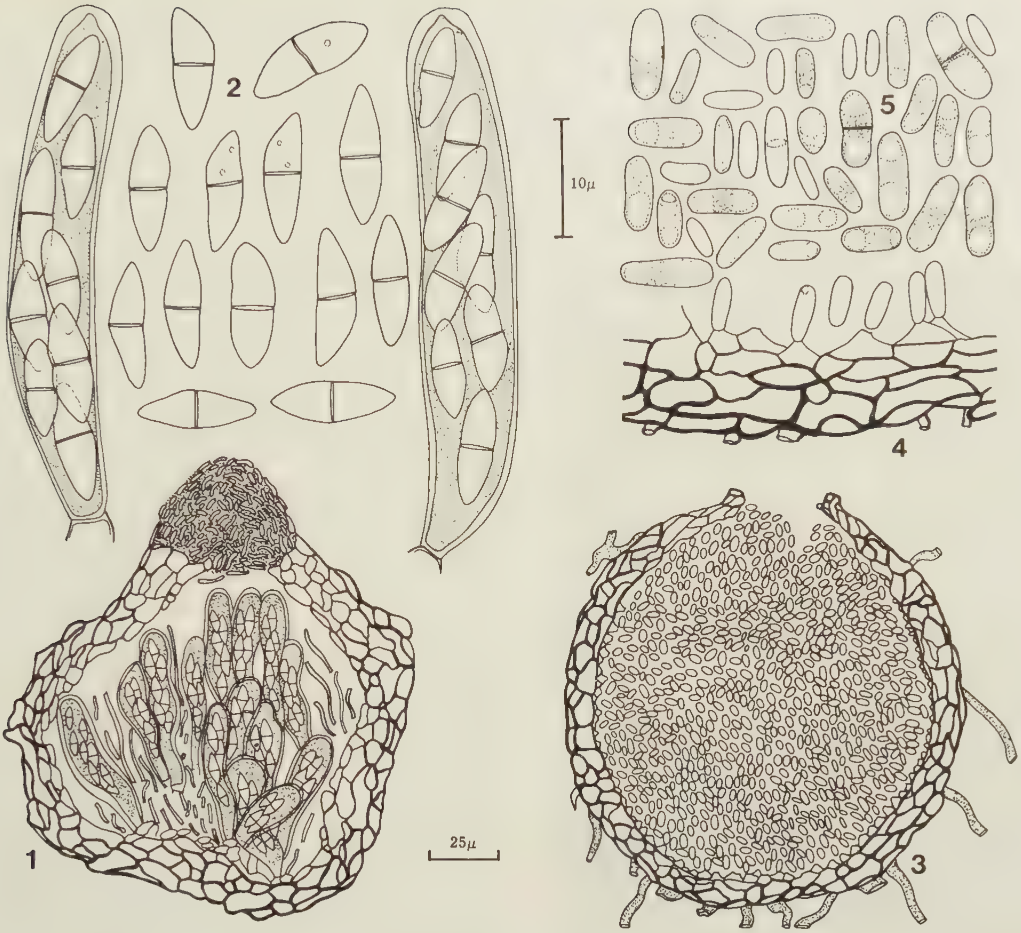


OCCURRENCE Phoma rot may be caused by several different fungal species. *Didymella lycopersici* (in its conidial state *Phoma lycopersici*) has been recorded on tomato fruits in the UK (6), Denmark, Romania, Morocco (1) and New Zealand (8), and on eggplant fruits in the USSR (7). *Phoma destructiva* occurs on tomatoes in the USA (9), India (4), the Pacific Islands (8), the UK (6), the USSR (7) and Italy, for example. It has also been recorded on peppers in the southern USA and on eggplants in Italy. *Phoma exigua* has been recorded on tomatoes in New Zealand (8) and on eggplants in India and Brazil (10). It can be difficult to distinguish between these three fungi, and they have often been confused (2,6,8).

SYMPTOMS Firm sunken lesions may occur on any part of the fruit but are most common at the stem-end. Infected tissue is at first brown and later black. Minute black bodies (pycnidia) are produced and project above the skin of the lesion; under humid conditions white or pinkish spores (conidia) ooze out (4,6,10). Microscopic examination may be necessary to distinguish phoma rot from pleospora rot (q.v.) in tomatoes and from phomopsis rot (q.v.) in eggplants (10).

BIOLOGY Phoma species persist in the soil and on crop debris, and can also survive on stakes used to support the crop (1). The sexual state (perithecia giving rise to ascospores) has been recorded for one of the species but occurs rarely (5). All three species exist in the asexual state (pycnidia giving rise to conidia) (2,6,8), and initial infection usually takes place when spores are splashed on to the plants by rain or irrigation water. Spores may also be transmitted in the recirculated solutions used in hydroponic culture (3). Lesions typically develop at wound sites on stems (3), but leaf-spotting is also reported on peppers and eggplants (7). Under moist conditions during pruning operations, rapid secondary spread can occur as exuded spores are transmitted on knives, hands and clothing (3). Infection of fruits usually takes place via cracks at the stem-end or through injuries, and can result in rapid rotting and fruit drop. Fruits infected shortly before harvest appear sound when packed, and lesions develop during transit and storage. Disease development is favoured by temperatures around 20°C (4) and decay is particularly rapid in ripe fruits (9). Seeds may become infected, thereby transmitting the disease to the next crop (5).

CONTROL If it has not been possible to sow clean seed in clean soil, then the developing seedlings should be drenched with a fungicide. If symptoms are noticed in the field or greenhouse, diseased plants (together with the soil around their roots) should be promptly and carefully removed for deep burial or burning. Pruning should be carried out only under dry conditions, and systematic sterilisation of knives can reduce the spread of disease. A fungicide can be used either to spray the plants or to drench the stem-bases and soil (10) or to 'paint' individual stem lesions (3). It is desirable to harvest the fruits dry, to handle them with care so as to minimise injury, and to ensure cool transit conditions followed by prompt ripening. In order to prevent the disease establishing itself in the next crop, it is essential to introduce strict hygienic practices promptly at the end of the season, including removal of plant debris followed by sterilisation of soil, stakes and greenhouse structures. Disinfection may be achieved with chemicals, steam or, in warm countries, by the use of solarization (page 132) (1).



82 *Didymella lycopersici* (from CMI Descr. No. 272, Holliday & Punithalingam 1970) 1 pseudothecium 2 asci and ascospores 3 pycnidium 4 part of pycnidial wall and conidiophores 5 conidia

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1 BESRI M. (1983) Lutte contre le chancre à *Didymella lycopersici* de la tomate par chauffage solaire (solarisation) des tuteurs. *Phytopathologische Zeitschrift* **108**, 333–340.

2 BOEREMA G.H. & DORENBOSCH M.M.J. (1973) The *Phoma* and *Ascochyta* species described by Wollenweber and Hochapfel in their study on fruit-rotting. *Studies in Mycology* **3**, 50 pp illus.

3 FAGG J. & FLETCHER J.T. (1987) Studies of the epidemiology and control of tomato stem rot caused by *Didymella lycopersici. Plant Pathology* **36**, 361–366.

4 HASIJA S.K. & BATRA S. (1979) Pathological studies on *Phoma destructiva* causing rot of tomato fruits. *Indian Phytopathology* **32**, 327–328.

5 HICKMAN C.J. (1946) Infection of outdoor tomato crops by *Didymella lycopersici. Journal of Pomology* **22**, 69–75.

6 KNIGHT D.E. (FISHER) (1960) Studies on *Didymella lycopersici* Kleb., the causal fungus of stem rot disease of tomatoes. *Transactions of the British Mycological Society* **43**, 519–522.

7 KOGAN E.D. (1977) [The pathogen of black rot of tomato fruits in Moldavia.] *Izvestiya na Moldavskogo SSR* (*Biologiya i Khimiya*) No. 2, 81–82.

8 LAUNDON G.F. (1971) Records of fungal plant diseases in New Zealand – 2. *New Zealand Journal of Botany* **9**, 610–624.

9 NIGHTINGALE A.A. & RAMSEY G.B. (1935) Development of Phoma rot of tomatoes in transit and in storage. *Circular of the United States Department of Agriculture* No. 371, 8 pp.

10 TERANISHI J. & FIGUEIREDO M.B. (1968) Podridões em frutos e hastes de beringela (*Solanum melongena* L.) causados por *Ascochyta phaseolorum* Sacc. *Biológico* **34**, 206–208.

PHOMOPSIS ROT of tomatoes, peppers and eggplants caused by

Diaporthe capsici **Punith.**
Conidial state: *Phomopsis capsici* (Magnaghi) Sacc.
Diaporthe phaseolorum **var.** ***sojae*** **(Lehman) Wehmeyer**
Conidial state: *Phomopsis phaseoli* (Desm.) Grove
Diaporthe vexans **(Sacc. & Sydow) Gratz**
Conidial state: *Phomopsis vexans* (Sacc. & Sydow) Harter

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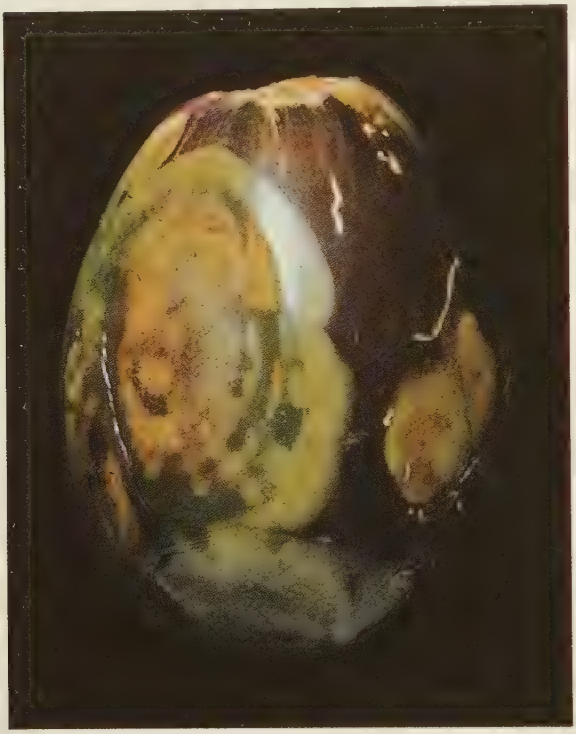
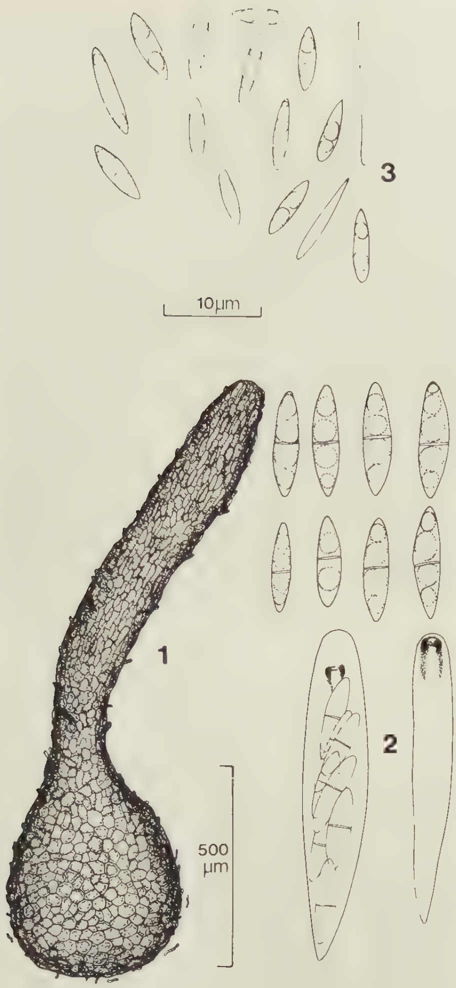


OCCURRENCE *D. capsici* has been recorded on peppers in Kenya, India, the Philippines and Fiji (9). *D. phaseolorum* var. *sojae* attacks tomatoes and peppers in the northern and southern USA (7,10), and losses in tomatoes have also been recorded in Hawaii and Trinidad. Phomopsis rot of eggplants, caused by *D. vexans* (5) occurs in the USA (6), Brazil (4), Puerto Rico (8), the Philippines (3), China, India (2), Mauritius (4), and other parts of Africa and Asia (CMI Map 329).

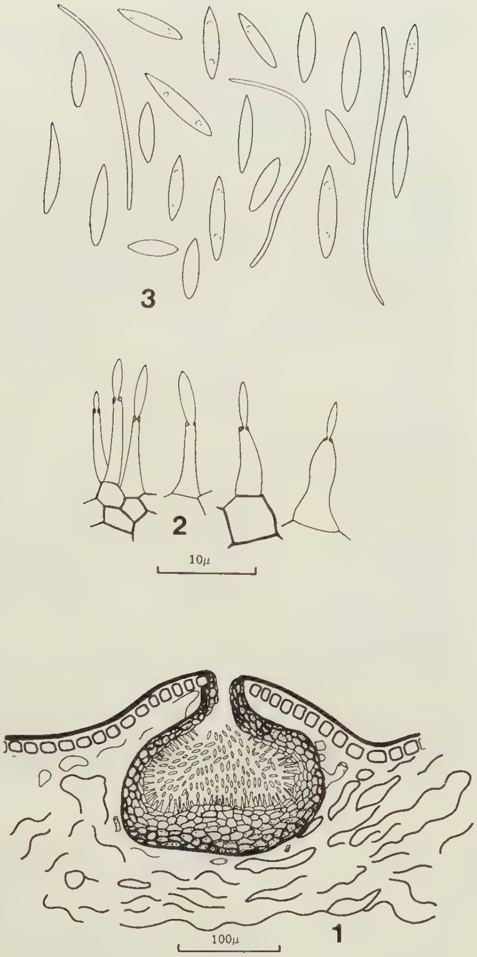
SYMPTOMS In tomatoes and peppers the lesions are at first soft and watersoaked and, under humid conditions, may support a white mould growth externally or (in peppers) on the inner wall (1,7,10). Later the infected tissue becomes tough, dark and shrivelled, as minute black bodies (pycnidia) develop, often in concentric zones. Lesions on eggplants are similar except that infected tissue is discoloured brown (4,8). Microscopic examination of the spores may be necessary to distinguish this disease from phoma rot of eggplants (q.v.) (6,8).

BIOLOGY The fungi which cause phomopsis rot occasionally produce the sexual state (perithecia giving rise to ascospores) (5,9,10). Usually, however, they survive in the asexual state (pycnidia giving rise to conidia) on weeds or on crop debris in the soil (1,4). During rain or irrigation exuded spores are splashed on to the plants and can cause infection of stems, leaves and fruits. The species which attack tomatoes and peppers are able to infect fruits only at weak points such as the blossom-end, sunscald lesions or wounds (7). Rotting is most rapid at temperatures between 25° and 30°C, but decay does not spread into adjacent healthy fruit during storage. The species attacking eggplants can cause devastating losses in the field. The fungus is able to penetrate healthy tissue directly, inducing stem blight, leaf-spotting, wilt and fruit-rotting (3,4,8). If fruits are infected shortly before harvest they may appear sound when packed, only to decay in store, the optimal growth temperature for the fungus being between 25° and 30°C.

CONTROL No specific measures have been devised for the control of phomopsis rot of tomatoes and peppers, but field sanitation and careful handling are likely to be helpful in reducing losses. With regard to phomopsis blight of eggplants, however, several control measures are available. A 3-year or 4-year crop rotation is beneficial; the fungus does not infect other crops, though it can survive on weeds of *Solanum* species, which should therefore be destroyed (4). It is desirable to obtain clean seed, since treatment of infected seed may be only partially effective (4). In addition it may be advisable to drench the seedbed with a disinfectant. The developing crop can be sprayed with a fungicide, although such treatment will not protect fruits in contact with infested soil (2). Diseased crop debris should be promptly removed and destroyed (8). Several eggplant cultivars have been bred for resistance to this important disease (3,4).



84 *Diaporthe capsici* (from CMI Descr. No. 733, Punithalingam 1982) 1 perithecium 2 immature and mature asci and ascospores 3 conidia



85 *Phomopsis vexans* (from CMI Descr. No. 338, Punithalingam & Holliday 1972) 1 pycnidium 2 conidiophores 3 conidia

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1 CEPONIS M.J. & FRIEDMAN B.A. (1955) The occurrence of Phomopsis on tomatoes in New York. *Plant Disease Reporter* **39**, 989–990.

2 DHARAM SINGH & CHAKRABARTI A.K. (1982) Chemical control of *Phomopsis* fruit rot of brinjal. *Indian Phytopathology* **35**, 314–315.

3 DIVINAGRACIA G.G. (1969) Histopathology of eggplant stems and leaves infected by *Phomopsis vexans*. *Philippine Agriculturist* **53**, 185–201.

4 FELIX S., LALLMAHOMED G.M. & ORIEUX L. (1965) Eggplant wilt *Diaporthe vexans* (Saccardo & Sydow) Gratz in Mauritius. *Revue Agricole et Sucrière de l'Île Maurice* **44**, 182–186 illus.

5 GRATZ L.O. (1942) The perfect stage of *Phomopsis vexans*. *Phytopathology* **32**, 540–542 illus.

6 HARTER L.L. (1914) Fruit rot, leaf-spot, and stem-blight of the eggplant caused by *Phomopsis vexans*. *Journal of Agricultural Research* **2**, 331–338 illus.

7 LUTTRELL E.S. (1947) *Diaporthe phaseolorum* var. *sojae* on crop plants. *Phytopathology* **37**, 445–465 illus.

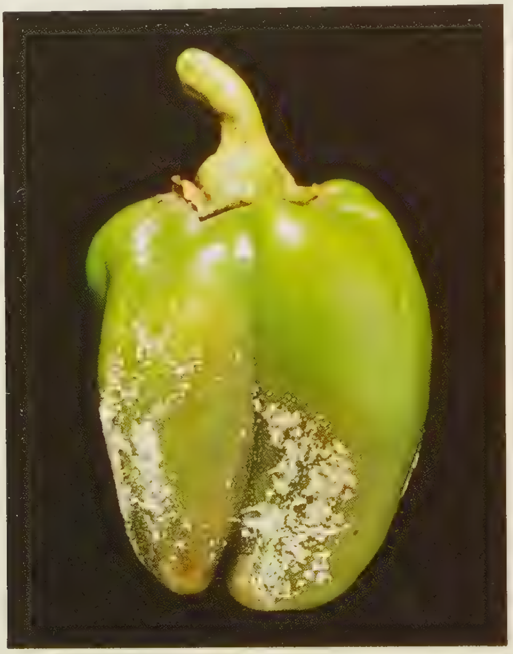
8 NOLLA J.A.B. (1929) The eggplant blight and fruit rot in Porto Rico. *Journal of the Department of Agriculture of Porto Rico* **13**, 35–57 illus.

9 PUNITHALINGAM E. (1981) Studies on Sphaeropsidales in culture. III. *Mycological Paper* No. 149, 42pp illus.

10 TUCKER C.M. (1935) *Diaporthe phaseolorum* on pepper fruit. *Mycologia* **27**, 580–585 illus.

PHYTOPHTHORA ROT of tomatoes, peppers and eggplants caused by ***Phytophthora* spp.**

86



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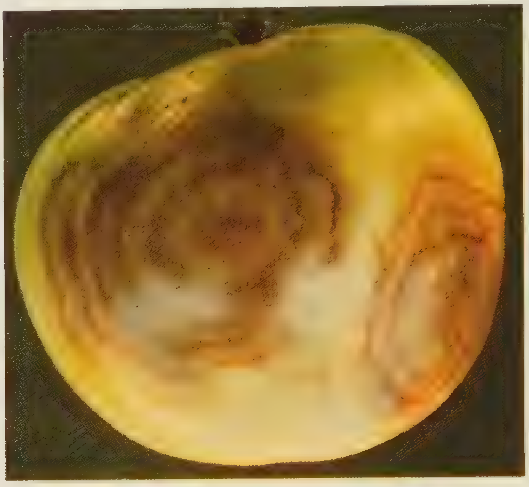

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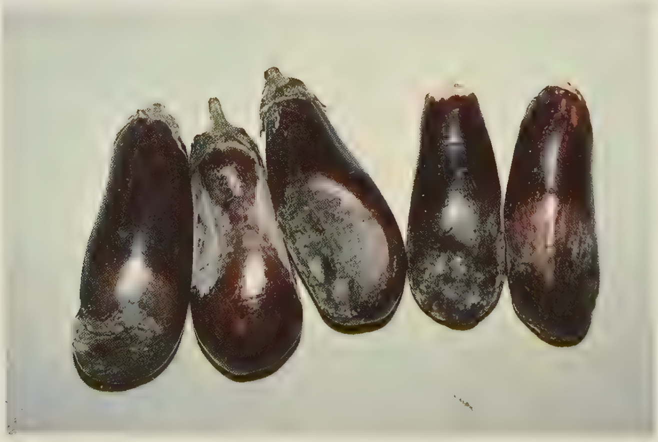
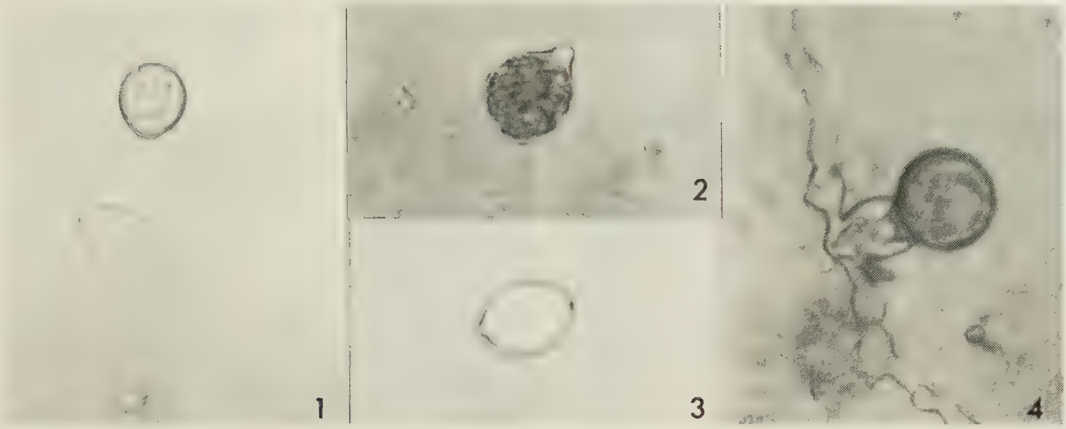


OCCURRENCE The disease occurs in both temperate and tropical countries, including the Netherlands (10), Italy (2), Greece (8), Bulgaria (3), the USA (4,6,9), Brazil (7), India (1,5), the Philippines and Japan. Both outdoor and greenhouse crops may be affected. The commonest species of *Phytophthora* on tomatoes, peppers and eggplants are *P. capsici* Leonian and *P. nicotianae* var. *parasitica* (Dastur) Waterh. Other records include *P. drechsleri* (page 39) and *P. palmivora* (Butler) Butler on tomatoes. Phytophthora rot of tomatoes is usually called 'buckeye rot', except when the causal fungus is *P. infestans* (page 309), in which case it is called late blight (q.v.).

SYMPTOMS Lesions on tomatoes begin as watersoaked spots which later develop concentric rings of various shades of brown (2). In peppers decay tends to start at the stem-end and the invaded tissue becomes dark green without any zonation. Lesions on eggplants are often near the distal end; they are dark brown with a conspicuous pale border. A humid atmosphere permits the development of an off-white mould bearing numerous spores (or, more strictly, sporangia).

BIOLOGY These fungi can survive in seed and soil, as thick-walled spores (oospores and, in some species, chlamydospores). Warm wet conditions



89 *Phytophthora nicotianae* var. *parasitica* (from CMI Descr. No. 35, Waterhouse & Waterston 1964) 1, 2, 3 sporangia 4 oogonium with amphigynous antheridium (stained with cotton blue in lactophenol) × 500

favour the production of thin-walled sporangia (1). These may in turn release swimming spores (zoospores), which may be splashed on to the aerial parts of the plant and are capable of causing direct infection of uninjured tissue. Fruits in contact with the ground are especially vulnerable (2,9). Decay continues after harvest, spreading into neighbouring healthy fruits.

CONTROL Crop rotation with non-susceptible crops helps to reduce disease incidence in the field. In greenhouses it is usually feasible to sterilise the soil before planting the crop (2). Clean or treated seed should be sown, and the developing crop may need to be protected with fungicide sprays (3). Trickle irrigation is preferable to sprinklers, and good drainage is especially desirable since persistently wet soil is conducive to disease (4). It is advantageous to stake the plants so as to prevent contact between fruits and soil. This may also be achieved by means of a plastic or straw mulch (9). Some resistant cultivars are available (7).

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2 D'ERCOLE N. (1980) *Phytophthora nicotianae* var. *nicotianae*, agente di marciume nel pomodoro in coltura protetta. *Informatore Fitopatologico* **30**(11/12), 21–24, 33, illus.
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4 HOY M.W., OGAWA J.M. & DUNIWAY J.M. (1984) Effects of irrigation on buckeye rot of tomato fruit caused by *Phytophthora parasitica*. *Phytopathology* **74**, 474–478.
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6 KREUTZER W.A. & BRYANT L.R. (1946) Certain aspects of the epiphytology and control of tomato fruit rot caused by *Phytophthora capsici* Leonian. *Phytopathology* **36**, 329–339 illus.
7 REIFSCHNEIDER F.J.B., CAFE-FILHO A.C. & REGO A.M. (1986) Factors affecting expression of resistance in pepper (*Capsicum annuum*) to blight caused by *Phytophthora capsici* in screening trials. *Plant Pathology* **35**, 451–456.
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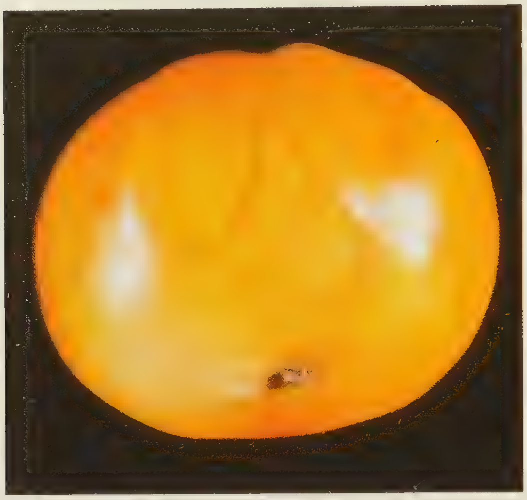
See also:

KIM C.H. (1989) Biological control of *Phytophthora* blight of red pepper in Korea. *Korean Journal of Plant Pathology* **5**, 100–105.

PINK MOULD ROT of tomatoes and eggplants caused by ***Trichothecium roseum*** **Link**

90



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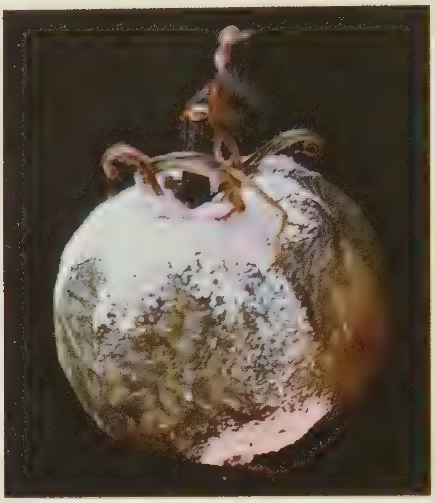

OCCURRENCE Pink mould rot is a minor disease of tomatoes, but occasional losses in the greenhouse crop have been reported from the USA (2,5), the UK (4), Italy (1) and Czechoslovakia (3). The disease has also been recorded on eggplants in the market in India.

SYMPTOMS Firm greyish-brown lesions are most often found at the blossom-end of affected fruits and, in the early stages, symptoms of this disease may resemble those of the physiological disorder blossom-end rot (q.v.) (5). However, lesions caused by the pink mould fungus possess a watersoaked margin and, under humid conditions, give rise to characteristic orange-pink spores (2). Lesions may occur on other parts of the fruit surface (4), and in tomatoes the fungus occasionally causes a stem-end rot, following cladosporium rot (q.v.) of the calyx.

BIOLOGY Pink mould can survive on plant debris and, under moist conditions, produces abundant asexual spores (conidia). If the prevailing weather is cool and damp, with overcast skies, tomato plants do not thrive, and there is an increased likelihood of infection by this fungus (5). Infection characteristically takes place via the dead flower parts which often remain attached to the developing fruits (5). Infected fruits may drop prematurely (3). Since eggplants too tend to rot from the blossom-end, it is probable that the mode of infection is similar. The fungus can also gain entry via wounds (2) or directly through the skin of fruits which have been stored at temperatures below about 10°C (4). Subsequent removal to 20°C (the optimal growth temperature of the fungus) permits rapid development of pink mould rot (2).

CONTROL In protected crops, improved ventilation of the greenhouse is beneficial (1), and it may be worthwhile to install fan heaters in order to maintain temperatures and humidities conducive to healthy plant growth. Infection and rotting can be minimised by prevention of injury to the fruits (2) and by the use of correct storage temperatures (4).

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1 CARTIA G. (1982) Marciume su pomodoro da *Trichothecium roseum. Informatore Fitopatologico* **32**(4), 21–22.

2 DEEMS R.E. (1951) Trichothecium fruit rot of glasshouse tomatoes. *Phytopathology* **41**, 633–640 illus.

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4 TOMKINS R.G. (1963) The effects of temperature, extent of evaporation and restriction of ventilation on the storage life of tomatoes. *Journal of Horticultural Science* **38**, 335–347.

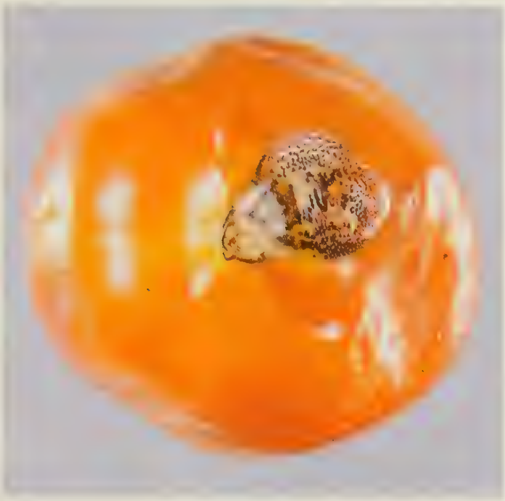
5 WELCH A.W., JENKINS S.F. & AVERRE C.W. (1975) Trichothecium fruit rot on greenhouse tomatoes in North Carolina. *Plant Disease Reporter* **59**, 255–257 illus.

PLEOSPORA ROT of tomatoes caused by

***Pleospora herbarum* (Pers.) Rabenh.**

Conidial state: *Stemphylium herbarum* Simmons

92



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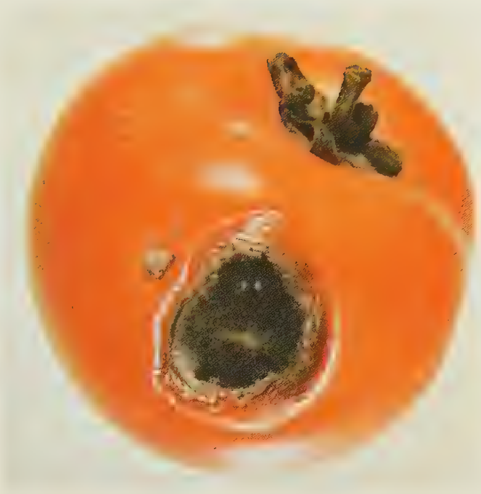

OCCURRENCE Pleospora rot has caused losses in tomatoes grown in the western and southern USA during the winter season (1,3,5). It has also been observed in Israel and the UK, and in consignments of Mexican tomatoes imported into the USA (5). Several species of *Stemphylium* are responsible for grey leaf spot of tomato in the USA, Israel and France (2).

SYMPTOMS Brown to black lesions form at the edge of the stem-scar or in wounds on any part of the fruit. Under humid conditions a greyish brown mould develops, giving rise to dark spores (conidia) (3) which are similar in general appearance to those of alternaria rot (q.v.). Mature lesions remain fairly firm, and are characterised by the production of minute black bodies (perithecia) (4) which protrude through the skin (5), conferring an appearance similar to that of phoma rot (q.v.). The identity of the fungus should therefore be confirmed by microscopic examination of the spores.

BIOLOGY This fungus commonly produces both the sexual stage (perithecia giving rise to ascospores) and the asexual stage (conidia), and infection of tomatoes is associated with cool wet conditions in the field (3,5). Infection occurs via growth cracks or wounds in the maturing fruit (1,3), and may not be evident at the time of harvest. Decay is favoured by storage temperatures around 20°C and proceeds more rapidly as the infected fruits ripen; the disease does not, however, spread into adjacent healthy fruits during storage (5).

CONTROL Tomatoes should be picked at the correct maturity so as to permit prompt and normal ripening. Careful handling is important, and fruits should be graded out if they exhibit injuries or incipient infections. Some tomato cultivars have been selected for their resistance to this disease (2).

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1 BARTZ J.A. (1972) Studies on the causal agent of black fungal lesions on stored tomato fruit. *Proceedings of Florida State Horticultural Society* **84**, 117–119.

2 BLANCARD D. & LATERROT H. (1986) Les Stemphylia rencontrés sur tomate. *Phytopathologia Mediterranea* **25**(1–3), 140–144.

3 BUTLER E.E. (1959) Fungi and rots in Californian canning tomatoes. *Plant Disease Reporter* **43**, 187–192.

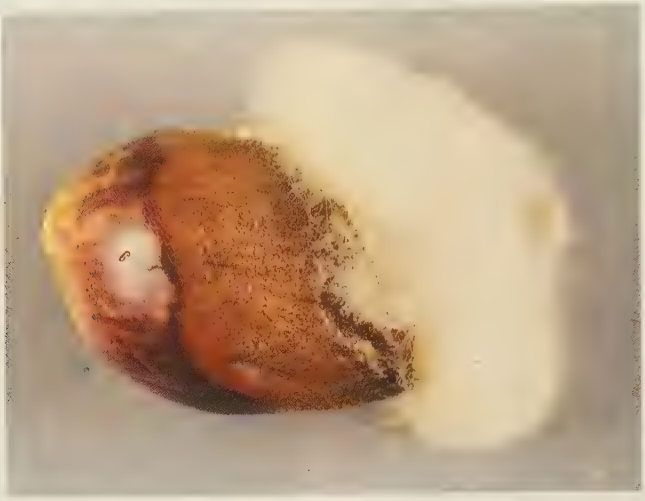
4 LEACH C.M. (1971) Regulation of perithecium development and maturation in *Pleospora herbarum* by light and temperature. *Transactions of the British Mycological Society* **57**, 295–315.

5 RAMSEY G.B. (1935) Pleospora rot of tomatoes. *Journal of Agricultural Research* **51**, 35–43 illus.

RHIZOPUS ROT of tomatoes, peppers and eggplants caused by

Rhizopus oryzae **Went & Prinsen Geerligs**
Rhizopus stolonifer **Lind**

94



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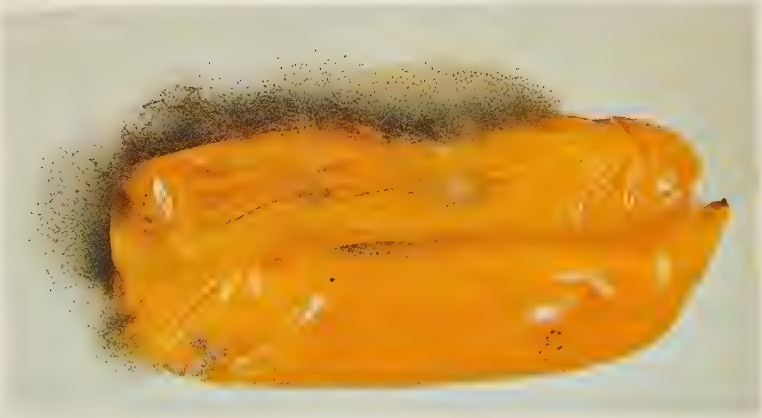
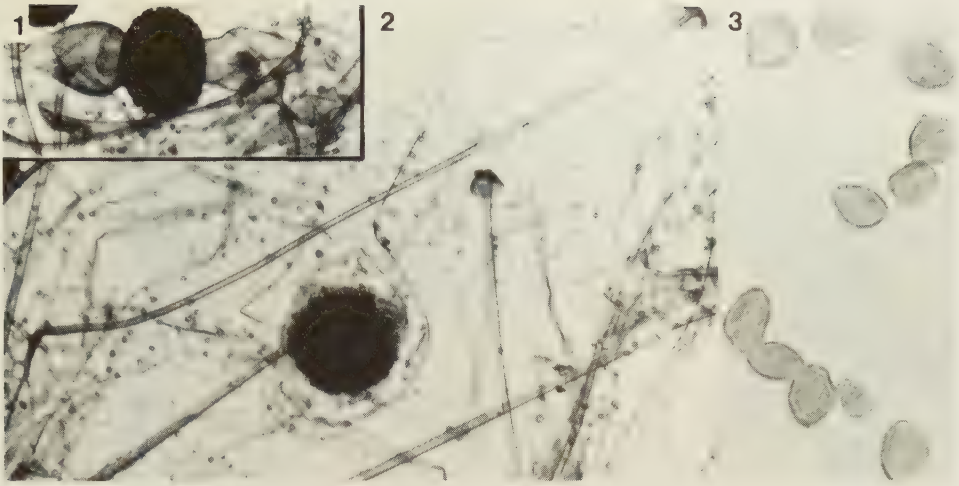


OCCURRENCE Rhizopus rot caused by *R. stolonifer* may occur in all countries producing tomatoes, peppers and eggplants, for example the USA (2,4,5,6,9), Israel (3), Romania (10), Japan (7) and India (8). *R. oryzae* (page 373) is common in tropical countries, for example Nigeria and India (1).

SYMPTOMS Lesions on tomatoes and peppers are soft and watersoaked but not discoloured. Decay spreads throughout the fruit, which often ruptures with much leakage of juice. There is an odour of fermentation, in contrast to the putrid smell associated with bacterial soft rot (q.v.). Subsequently a coarse white mould appears, bearing globular spore-heads (sporangia) which turn from white to black (5). In eggplants the lesions are soft and brown and, under humid conditions, support copious mould growth as described above (1).

BIOLOGY These fungi are very common in soil and in the atmosphere (9). Occasionally *R. stolonifer* produces the sexual state (zygospores) (2) and *R. oryzae* may produce resting spores (chlamydospores). However, both fungi more commonly exist in the asexual state (sporangia giving rise to sporangiospores); in a warm moist environment these spores germinate to give mould strands. Initial infection occurs only via injuries (3,5). Maturing tomatoes may develop growth cracks, in which fruit flies lay their eggs and at the same time transmit fungal spores which later cause infection and decay (4). Both green and ripe tomatoes may undergo rotting (9). Field infection has also been recorded in peppers (7) and eggplants (1). Infection frequently occurs in fruits damaged by rough handling during and after harvest, especially if field boxes and packing lines are allowed to become contaminated (2,9). Once either of these fungi is established in a fruit,



96 *Rhizopus stolonifer* (from CMI Descr. No. 524, Lunn 1977) 1 zygospore formed between two compatible isolates × 100 2 sporangium, sporangiophores and rhizoids × 60 3 sporangiospores × 1000

decay can spread into adjacent sound fruits, causing extensive 'nesting' (3,5). The optimal growth temperature for *R. stolonifer* is approximately 25°C and for *R. oryzae* 35°C.

CONTROL An effective means of reducing losses is to improve standards of hygiene in field and packing house (2). Fallen or culled fruits should not be left to rot, since many spores would be released into the air or disseminated by fruit flies attracted by the odour of decay (4). Field boxes should be regularly disinfested by heat treatment, and care should be taken to remove all sharp projections from containers and grading machinery (2,9). Post-harvest treatments to control the spread of rhizopus rot include hot-water baths, irradiation, fungicide dips, waxing, and the use of paper wraps impregnated with fungicide (5,6,8). Refrigerated storage is beneficial, since these fungi grow slowly at temperatures around 10°C (7,8).

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1 ALI S.S. & SHUKLA A. (1981) Pathogenesis of *Rhizopus oryzae* Went & Geerligs in fruit-rot disease of brinjal (*Solanum melongena* L.). *Current Science* **50**, 232–233.
2 BAKER K.F. (1946) An epiphytotic of Rhizopus soft rot of green-wrap tomatoes in California. *Plant Disease Reporter* **30**, 20–26.
3 BARKAI-GOLAN R. & KOPELIOVITCH E. (1981) Resistance of *rin* and *nor* tomato mutants to postharvest *Rhizopus* infection. *Annals of Applied Biology* **98**, 289–293.
4 BUTLER E.E. & BRACKER C.E. (1963) The role of *Drosophila melanogaster* in the epiphytology of *Geotrichum, Rhizopus,* and other fruit rots of tomato. *Phytopathology* **53**, 1016–1020.
5 ECKERT J.W. & KOLBEZEN M.J. (1962) Controlling spread of *Rhizopus* in packed tomatoes by tissue wraps impregnated with active halogen compounds. *Phytopathology* **52**, 180–183 illus.
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7 KURATA M., KOTANI S., YAMAMOTO L. & SAITO M. (1975). [A new postharvest decay of pepper caused by *Rhizopus nigricans* Ehr.] *Bulletin of Kochi Prefectural Institute of Agricultural and Forest Science* **7**, 15–20.
8 ROY M.K. (1981) Radiation, heat and chemical combination in the control of *Rhizopus nigricans* in tomato fruits. *Journal of Nuclear Agriculture and Biology* **10**, 8–9.
9 SONODA R.M., HAYSLIP N.C. & STOFFELL P.J. (1981) Tomato fruit rot infection cycle in a fresh market packing operation. *Proceedings of Florida State Horticultural Society* **94**, 281–282.
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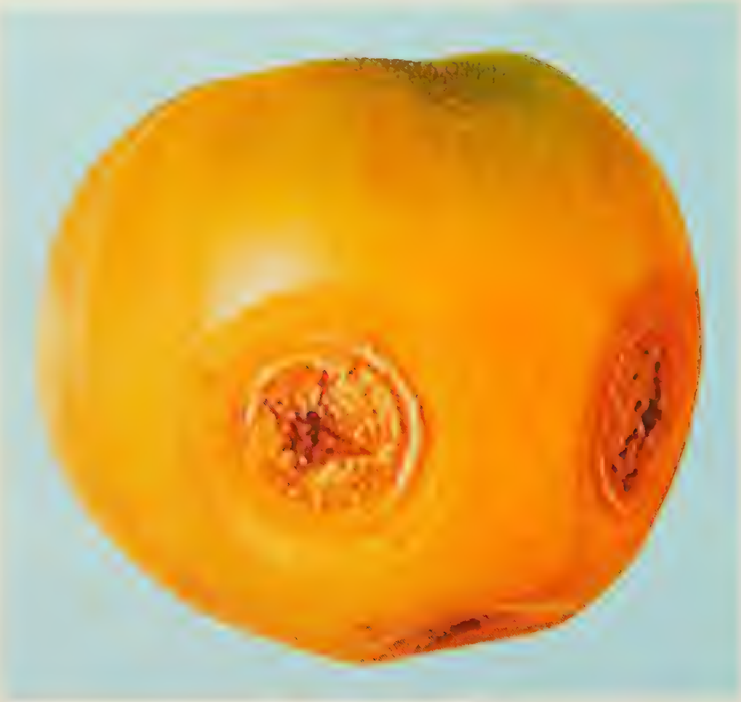
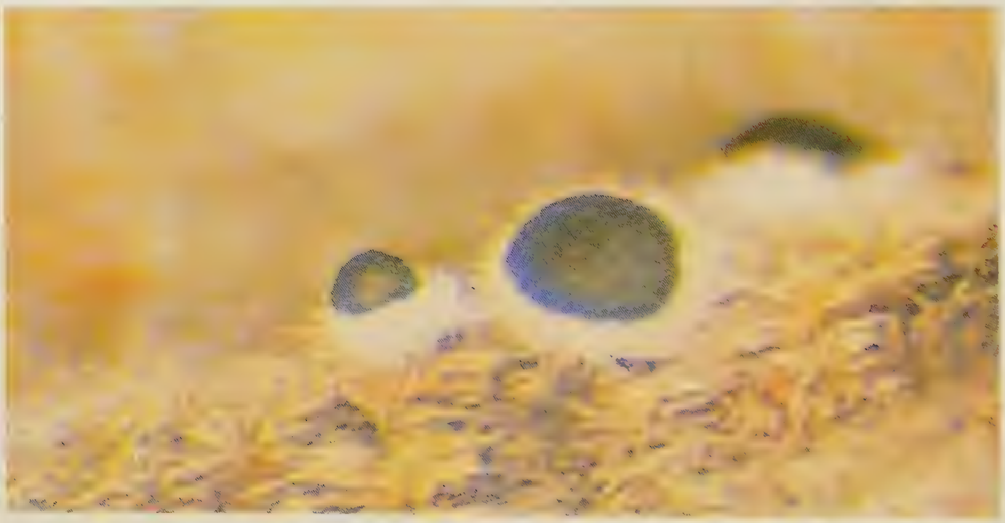
RING ROT of tomatoes, peppers and eggplants caused by ***Myrothecium roridum*** **Tode ex Fr.**

97

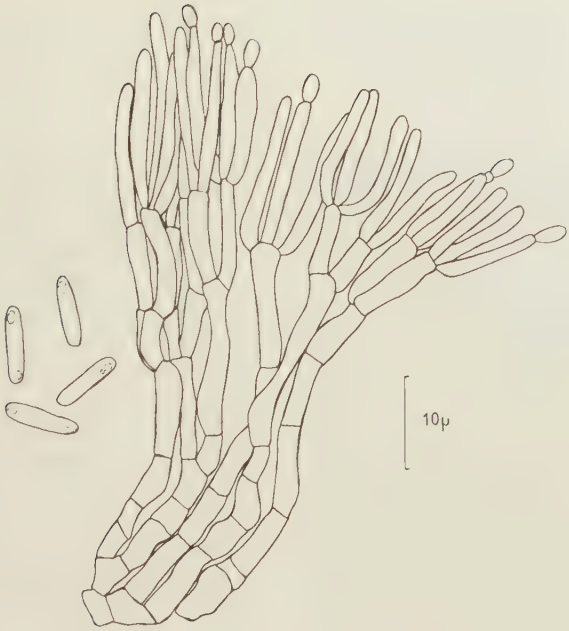




98 Sporodochia of *Myrothecium roridum*

OCCURRENCE Ring rot may cause lesions in outdoor and greenhouse crops. *M. roridum* has been recorded on tomatoes from Cuba, Mexico and the USA (7). In India it may attack tomatoes (1,8), chilli peppers (2) and eggplants (10). A related but less virulent fungus, *M. verrucaria* (CMI Descr. 253), has been observed on tomatoes in the USA (3,7,9).

SYMPTOMS Lesions are circular to oval, firm and slightly sunken, with a sharply defined margin. They are characterised by narrow concentric zones of white mould alternating with moist dark green to black spore masses (8,10). Decay extends deep into the fruit, and the corky rotted tissue can be lifted out with ease (7).



99 *Myrothecium roridum* (from CMI Descr. No. 253, Fitton & Holliday 1970) Conidiophores and conidia

BIOLOGY The fungus survives on plant debris in the soil and, during warm wet weather, produces abundant asexual spores (conidia) (4). Dissemination of spores is thought to be by wind, water and insects, and leaf-spotting is a common symptom in many crops (3,5). Infection of fruits is facilitated by wounding (8,10), and decay is especially rapid in ripe fruits. Fungal growth and spore production are favoured by temperatures around 25°C; slow decay occurs at 10°C.

CONTROL Seed transmission has been reported in some crops (3), so a pre-planting treatment may be advisable. Some fungicides have proved effective against this fungus (6). Care should be taken to minimise mechanical damage to fruits during harvesting (8,10). Field hygiene is important, and crop debris should be promptly removed and destroyed. A few eggplant cultivars are resistant to the disease (5), and some peppers are immune (3).

1 GAUR S.C. & PATHAK V.N. (1979) Physiological studies on *Myrothecium roridum* inciting fruit rot of tomato. *Indian Journal of Mycology and Plant Pathology* **9**, 109–110.

2 LAXMINARAYANA P. & REDDY S.M. (1978) Some new post-harvest diseases of chillies. *Indian Journal of Mycology and Plant Pathology* **8**, 202–203 illus.

3 NGUYEN T.H., MATHUR S.B. & NEERGAARD P. (1973) Seed-borne species of *Myrothecium* and their pathogenic potential. *Transactions of the British Mycological Society* **61**, 347–354 illus.

4 PRESTON N.C. (1961) Observations on the genus *Myrothecium* Tode. 3. The cylindrical-spored species of *Myrothecium* known in Britain. *Transactions of the British Mycological Society* **44**, 31–41 illus.

5 SINGH D.V., KALDA T.S., SETH M.L. & CHOUDHURY B. (1972) Note on the resistance to *Myrothecium* blight in brinjal (*Solanum melongena* L.). *Indian Journal of Agricultural Sciences* **42**, 431–432.

6 SOHI H.S. & KALRA J.S. (1982) *In vitro* evaluation of different fungicides against *Myrothecium roridum*. *Pesticides* **16**(3), 20–21.

7 STEVENSON J.A. & McCOLLOCH L.P. (1947) *Myrothecium* as a tomato fruit rot organism. *Plant Disease Reporter* **31**, 147–149.

8 TANDON R.N. & SRIVASTAVA M.P. (1963) *Myrothecium* rot of tomatoes. *Current Science* **32**, 426–427 illus.

9 TULLOCH M. (1972) The genus *Myrothecium* Tode ex Fr. *Mycological Paper* No. 130, 42 pp illus.

10 ULLASA B.A., MAHOLAY M.N. & SOHI H.S. (1976) Ring rot of brinjal caused by *Myrothecium roridum* Tode ex Fries from Bangalore. *Current Science* **45**, 601–602 illus.

SCLEROTIUM ROT of tomatoes, peppers and eggplants caused by

Corticium rolfsii **Curzi**

Sclerotial state: *Sclerotium rolfsii* Sacc.

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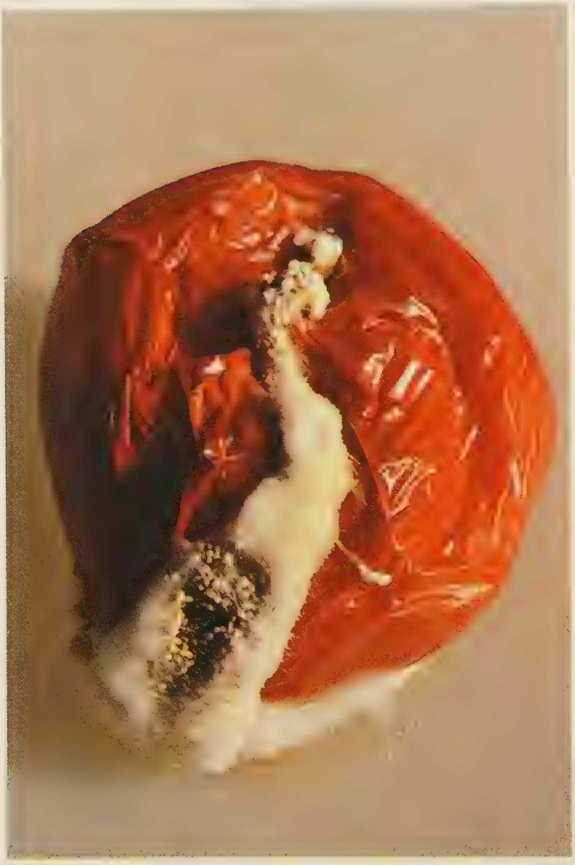

SYMPTOMS Decayed areas of the fruit are discoloured and slightly sunken, and the skin often splits. A bright white silky mould develops, and spreads in a characteristic fan-like formation over the decayed tissue. Numerous spherical bodies about 1–2 mm in diameter (sclerotia) develop on the mould; these are white at first, and later shrink slightly and become brown to dark brown (3).

BIOLOGY The fungus persists in the soil as resting bodies (sclerotia) and only rarely produces the sexual stage (a hymenium giving rise to basidiospores). In warm wet soil strands of mould grow out from sclerotia and, besides being capable of causing 'stem blight', the fungus can cause direct infection of sound fruits in contact with the ground. Fruits hanging close to the ground may become infected as a result of soil particles being splashed up during rain or irrigation. If fruits are kept at high temperatures after harvest, sclerotium rot develops rapidly and spreads into healthy fruits in contact. The optimal temperature for decay is approximately 30°C (3).

CONTROL Crop rotation is of limited value because so many crops are susceptible to attack (3). However, it can be advantageous to grow onions, which seem to reduce the viability of sclerotia (5). These resistant bodies have been known to survive in the soil for at least four years and are difficult to kill. Soil fumigation or drenching can be effective, followed by the addition of a particular fungus which is antagonistic to the sclerotia but otherwise harmless. It is advisable to stake the plants or to cover the soil with plastic sheeting, as a means of preventing contact between soil and fruits (1,2,4). Any diseased plants should be promptly and carefully removed, together with the surrounding soil, for immediate destruction (3). Post-harvest fruit rotting can be controlled by the use of refrigeration, since the fungus grows very slowly at 10°C and below (3).

OCCURRENCE The fungus is widespread in tropical, subtropical and warm temperate countries, and causes the field disease known as 'southern blight' in numerous crops. Sclerotium rot of tomato fruits has been recorded in the Philippines (3), Papua New Guinea (2), Nigeria, Israel (5) and the southern USA (1,4). Sclerotium rot of peppers occurs occasionally in the USA, and sclerotium rot of eggplants has been recorded in Greece.

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1 BROWN J.E., STEVENS C., OSBORN M.C. & BRYCE H.M. (1989) Black plastic mulch and spunbonded polyester row cover as method of southern blight control in bell pepper. *Plant Disease* **73**, 931–932.

2 DODD J. (1979) Effects of pests and diseases on the yield and quality of tomatoes in the Port Moresby area, Papua New Guinea. *Papua New Guinea Agricultural Journal* **30**, 53–59.

3 FAJARDO T.G. & MENDOZA J.M. (1935) Studies on the *Sclerotium rolfsii* Sacc. attacking tomato, peanuts, and other plants in the Philippines. *Philippine Journal of Agriculture* **6**, 387–424 illus.

4 JONES C.W. & McCARTER S.M. (1974) Etiology of tomato fruit rots and evaluation of cultural and chemical treatments for their control. *Phytopathology* **64**, 1205–1208.

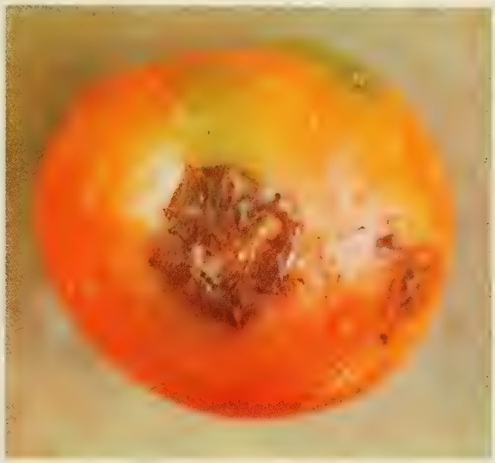
5 ZEIDAN O., ELAD Y., HADAR Y. & CHET I. (1986) Integrating onion in crop rotation to control *Sclerotium rolfsii*. *Plant Disease* **70**, 426–428.

SOIL ROT of tomatoes, peppers and eggplants caused by
***Thanatephorus cucumeris* (Frank) Donk**
Sclerotial state: *Rhizoctonia solani* Kühn

OCCURRENCE Soil rot is of particular importance on tomatoes, and heavy losses have been reported, both on fresh market fruit (1) and in consignments intended for processing (3). Most reports are from the southern USA, but the disease occurs in many other countries, including Israel (5), India, Iran and Brazil (4). Peppers and eggplants are occasionally affected.

SYMPTOMS On mature-green or pink tomatoes lesions are roughly circular with dark brown borders and pale centres. Concentric rings of different shades sometimes develop, and as the affected area enlarges it becomes slightly sunken, while still remaining firm. The skin may split in several directions, and rotting is rapid under warm conditions. In ripe tomatoes lesions tend to be uniformly brown or dark red, enlarging to an irregular shape. Infected flesh is soft and watery. A white or greyish-brown mould may be apparent on the surface of the fruit and on paper wraps used in packing (1). Soil rot lesions are occasionally found on peppers and eggplants (1).

BIOLOGY The fungus is very common in soil, and produces both the sexual state (a hymenium giving rise to basidiospores) and the sterile state (sclerotia). Under wet conditions the mould can attack fruits in contact with the ground, by direct penetration or via injuries (2,5). Less frequently infection may occur as a result of soil being splashed on to low-hanging fruits (1) or alternatively from airborne spores (3). The disease is especially common in tomatoes intended for processing, since these crops are often grown without the aid of stakes (2), and many fruits are therefore on or near the soil for an extended period (fruit intended for processing is left on the plant until fully ripe). Even in comparatively dry seasons the moisture retained by bushy plants can be sufficient to permit infection (2,3,5). The mould can spread into healthy fruits in contact (5), and at temperatures around 25°C rotting is extremely rapid.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CONTROL The fungus may be transmitted in seed, necessitating seed-treatment before planting (1); this is especially so in peppers. Weeds together with debris from previous infected crops should have been promptly and carefully removed. The incidence of soil rot can be reduced by staking the plants or by covering the soil with plastic sheeting; however, this precludes the possibility of mechanical harvesting, which is used for crops destined for processing. Fungicide sprays give partial control (4), and there have been promising results with biological control, in which an antagonist is added to the soil (5). Application of ethephon (an ethylene-producing compound) to the maturing crop has two advantages; firstly, it promotes uniform fruit ripening which permits prompt harvesting, and secondly it causes leaf drop, resulting in a reduction of humidity through increased local air movement (2). Efforts are being made to breed new cultivars resistant to the disease.

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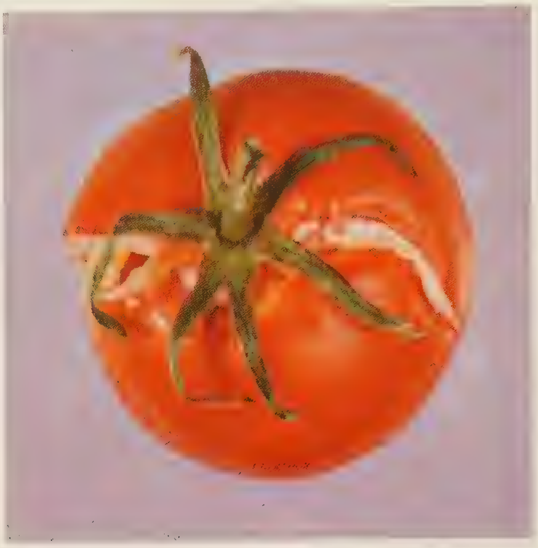
SOUR ROT of tomatoes caused by
Geotrichum candidum Link ex Pers.

102



OCCURRENCE Sour rot of tomatoes is common in the western and southern USA (1,3,4,5) and has also been recorded in Italy, Israel, Egypt (2), India and Nigeria.

SYMPTOMS On mature-green fruits the lesions appear pale, dull and watersoaked, and have a definite sour odour. On ripe fruits infected tissue is dark, soft and watery, and the sour odour may be superseded by the foul odour of bacterial soft rot (q.v.). Sour rot usually starts at the stem scar or at a wound, and if the skin splits then a creamy white mould develops on the exposed flesh, producing sticky spores (1,4). A closely related fungus, *G. penicillatum* (do Carmo-Sousa) v. Arx, causes decay without a sour odour (3).

BIOLOGY *G. candidum* is a common soil inhabitant in most parts of the world. Its spores (conidia) may be transmitted in air, water and, in particular, by insects. Flies visiting split and rotten fruits in a ripening crop can transfer spores to healthy fruit, and also cause wounds which permit infection (1). Fruits infected shortly before harvest may inadvertently be packed, and undergo decay during transit or storage. The fungus appears to thrive under conditions of poor ventilation, when the air is warm, moist, and low in oxygen (5). The optimal growth temperature for the fungus is approximately 30°C, but it can cause a very slow decay even at 2°C (4).

CONTROL It is important to control the insects which disseminate spores and puncture the fruit (1). Care should be taken to minimise mechanical damage during harvesting, and to discard fruit possessing stem-scar cracks. Post-harvest treatments include dipping the fruit in a fungicide or incorporating a suitable chemical in paper wrappers. Only a few fungicides are effective against the sour rot fungus (2). Different chemicals are required for the control of *G. penicillatum* (3).

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1 BUTLER E.E. (1961) Transmission of *Geotrichum* rot of tomato fruit by *Drosophila melanogaster. Phytopathology* **51**, 250–255 illus.

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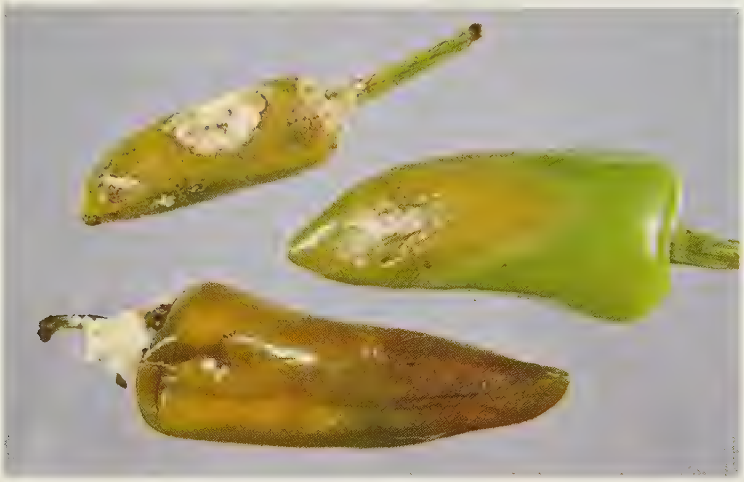
WATERY SOFT ROT of tomatoes, peppers and eggplants caused by

Sclerotinia minor **Jagger**
Sclerotinia sclerotiorum **(Lib.) de Bary**



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OCCURRENCE Watery soft rot occurs in temperate regions and in subtropical countries possessing a cool wet season. Records include the UK, France (1), Germany (2), Italy (3), Cyprus, Israel, parts of the USA (4), Australia, New Zealand (5) and Japan. Sclerotinia disease is frequently found in conjunction with grey mould rot (q.v.) (2).

SYMPTOMS Lesions may occur anywhere on the fruit, which, under warm moist conditions, is rapidly enveloped in white mould. Irregularly shaped bodies (sclerotia) develop on the mould; these are at first white, exuding drops of liquid, and later black, dry and hard. Those of *S. minor* are 1 to 2 mm in size, while those of *S. sclerotiorum* are 3 to 10 mm or more (5). Infected fruit tissue is soft and watery.

BIOLOGY These fungi can persist in the soil for many years as resting bodies (sclerotia), which 'germinate' after a period of rain or irrigation. Those of *S. minor* typically produce mould strands (5) which attack the stems at ground level. Those of *S. sclerotiorum* produce the sexual stage (apothecia giving rise to ascospores); the spores are ejected into the air and on to stems, fruits and foliage (2,5). Initial infection by this species is characteristically via dead flowers (4), from which the fungus can progress into developing fruits. If infected fruits are placed in store, decay spreads into adjacent healthy fruits, causing 'nesting'.

CONTROL Soil can be steam-sterilised or treated with chemicals in order to kill the sclerotia. For control of *S. sclerotiorum*, greenhouses can be made from special ultra-violet absorbing film which inhibits spore production by the fungus (2). Crops may be protected by fungicide sprays (3,5). Weeds should be controlled, since they may serve as alternative hosts for these fungi and also provide a shaded and humid environment conducive to disease development. Any infected plants should be promptly and carefully removed and burned, so as to kill all sclerotia.

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4 PURDY L.H. & BARDIN R. (1953) Mode of infection of tomato plants by the ascospores of *Sclerotinia sclerotiorum. Plant Disease Reporter* **37**, 361–362.

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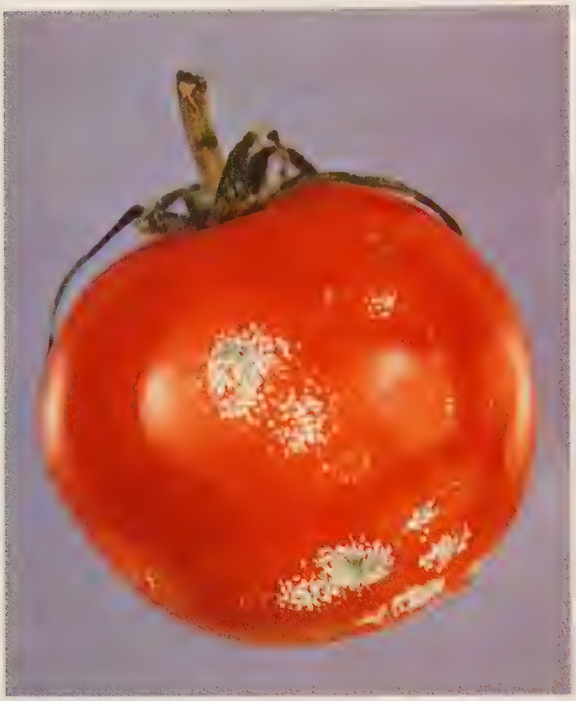


104 Blue mould rot

ASPERGILLUS ROT may be caused by any of several species, according to the temperature and humidity of the storage environment (10). *Aspergillus flavus* Link: Fr. and *A. niger* (page 242) have been recorded on tomatoes in the southern USA. A range of species, including *A. flavus* and *A. niger*, has been found on Indian chilli peppers. Invasion occurs via natural openings (stomata) in the stalks, and also via wounds in the skin of fruits; under dry conditions there may be no external mould growth but the internal wall of the peppers is colonised (10). *A. flavus* may produce a toxic substance (hence called aflatoxin) which makes infected produce unfit for consumption. *A. niger* causes a stylar-end rot of eggplants in India (4).

BLUE MOULD ROT is caused by species of *Penicillium*, for example *P. cyclopium* Westling, *P. expansum* Link and *P. italicum* (page 245). Fruits are predisposed to invasion if they have been weakened by chilling injury (q.v.), overlong storage in a modified atmosphere, or mechanical damage.

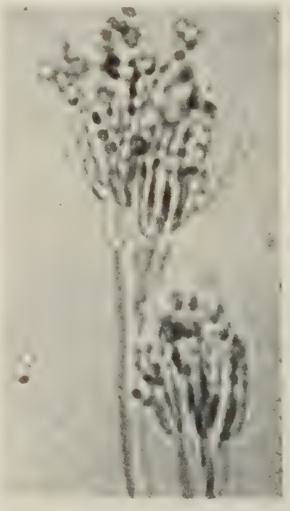
BOTRYODIPLODIA ROT has been recorded on tomatoes in the West Indies (1) and Nigeria, and on sweet peppers, chillies and eggplants in India. The most common species is *Botryodiplodia theobromae* (page 371), causing a soft rot which develops rapidly at tropical temperatures. It may be transmitted in seed.

CHARCOAL ROT has been reported on sunscorched tomato fruits in Nigeria (8) and on ripe pepper fruits in the USA (6) and in India, the causal fungus being *Macrophomina phaseolina* (page 25). In peppers the fungus may develop internally, colonising the seeds and only later becoming apparent externally when the skin darkens.

CHOANEPHORA ROT has caused losses of chilli peppers in India (3). Ripe fruits undergo a soft wet decay and become covered with characteristic spores of the fungus *Choanephora cucurbitarum* (page 27). The disease can be controlled by fungicide sprays (3).

CORYNESPORA ROT occurs occasionally on eggplants in India and on tomatoes in India, Cuba and the southern USA (2). The fungus, *Corynespora cassiicola* (Berk. & Curt.) Wei more commonly causes leaf-spotting, but fruits are occasionally infected, especially during warm wet weather. Lesions are at first dark brown and sunken, and later each develops a characteristic white centre. Decay is rapid, and causes premature ripening.

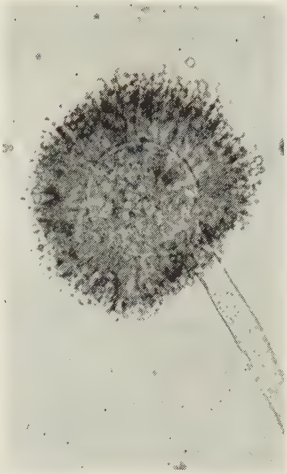
CURVULARIA ROT is caused by *Cochliobolus lunatus* Nelson & Haasis (conidial state: *Curvularia lunata* (Wakker) Boedijn). The disease has been recorded on tomatoes in the southern USA, India and Nigeria, and it is reported that sunscorched fruits are especially susceptible (8). The same fungus causes a dry rot of eggplants in India (4).



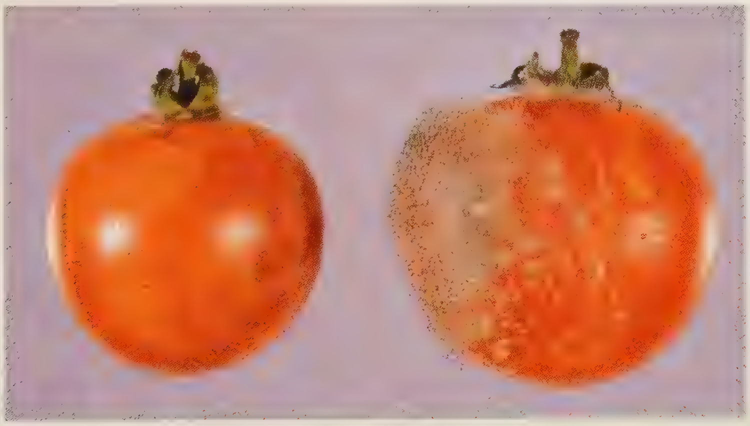
105 *Penicillium expansum*
(from CMI Descr.
No. 97, Onions 1966)
Conidial heads × 500

HELMINTHOSPORIUM ROT occasionally affects tomatoes, peppers and eggplants stored under warm conditions, especially if the fruits have previously been weakened in some way. *Helminthosporium carposaprum* Pollack caused losses in tomatoes from Mexico, after an unusual frost during the growing season. Sunken brown lesions developed and, under warm humid conditions, dark spores (conidia) were produced (5). It has been suggested that this fungus may be synonymous with *Corynespora cassiicola* (q.v.). In India a similar rot occurs on chillies and sweet peppers (9), and a species of *Helminthosporium* has consistently been found in seeds of eggplant.

MUCOR ROT may be caused by various species, including *Mucor mucedo* L. ex Fr. and *M. piriformis* Fischer. The moulds are white with black sporeheads, and they cause a soft wet rot. *M. mucedo* is particularly aggressive and at 25°C can cause complete decay of tomato fruits within 2 or 3 days (7).



106 *Aspergillus flavus* (from CMI Descr. No. 91, Onions 1966) Conidial head × 250



107 Mucor rot

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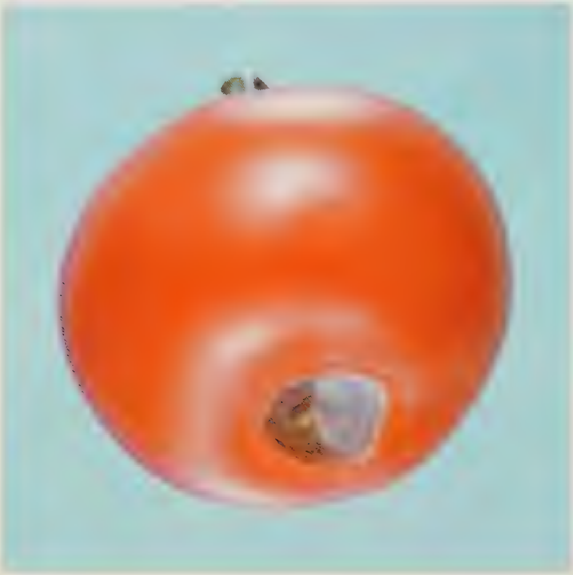
BLOSSOM-END ROT of tomatoes, peppers and eggplants

108



OCCURRENCE This is essentially a pre-harvest problem and occurs commonly, both in outdoor and in protected crops. Cultivars with large or elongated fruits are especially susceptible. A considerable amount of research has been carried out, for example in the UK (1), the USA (2,4), Canada (5) and Egypt (3). The term 'rot' is unfortunate, since no pathogenic organism is involved, it being a physiological disorder.

SYMPTOMS Affected fruits show a discoloured, leathery area at the distal end, pale in peppers, dark and sunken in tomatoes (4,5). If the fruit is cut, discoloured strands are sometimes visible in the tissue surrounding the seeds. The symptoms do not progress further after harvest, but nevertheless constitute an important market blemish (5).

BIOLOGY The disorder is initiated during the early stages of fruit development (3), and affected fruits may drop prematurely (4). More usually, however, the fruits continue to grow but are seriously disfigured. The cause is a local deficiency of calcium in the fruit tissue. During periods of rapid fruit growth the uptake and distribution of calcium may be hampered by water stress or by inappropriate use of fertilisers (4).

CONTROL It is important to ensure adequate water availability during early fruit development; in dry seasons frequent irrigation can be very effective in reducing the incidence of blossom-end rot (5). Calcium salts may be added to the soil or growth medium, or sprayed on to young plants. Reductions in application of other minerals can promote uptake of calcium (2). Such measures sometimes provide only partial control, however, the explanation being that most of the calcium is translocated to leaves rather than to fruits (1,2). In the controlled environment of the glasshouse, the following strategy may be used. During the daytime (when stomata are open for photosynthesis), measures should be taken to ensure a low atmospheric humidity. This encourages transpiration from the leaves and a corresponding uptake of water and minerals through the roots. This on its own is of little benefit to the fruits which, having no stomata, do not transpire. Thus a second requirement is a high humidity during the night; this induces a strong root pressure, causing the sap (with its calcium) to move from stems and leaves into the fruits (1). Healthy roots and an adequate water supply are obvious prerequisites.

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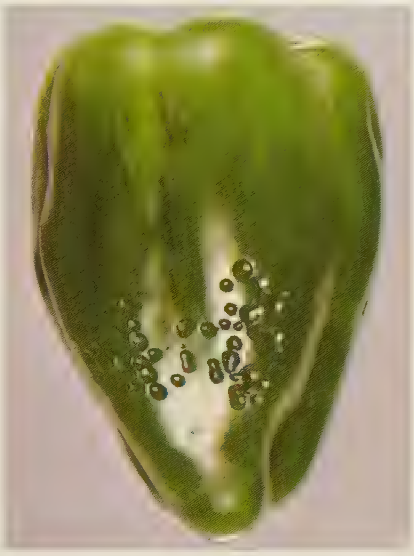
CHILLING INJURY of tomatoes, peppers and eggplants

OCCURRENCE This disorder is frequently encountered but not always correctly diagnosed. Research has been carried out in several countries, including the UK (2), the USA (1,4,5), Israel, Australia, China and Japan (3).

SYMPTOMS Typical symptoms are surface pitting, premature loss of firmness, and a markedly increased tendency to decay. Alternaria rot and cladosporium rot (q.v.) are often associated with chilling injury. Expression of symptoms is usually delayed until after the fruits are returned to warmer conditions. Tomatoes fail to ripen properly and show poor colour development (2). Severely damaged 'turning' tomatoes may remain abnormally firm owing to complete disruption of the ripening process.

BIOLOGY Sensitivity to chilling is influenced by several factors, including cultivar and growing conditions. In a study of eggplants (3) it was found that those grown out of doors were less susceptible than those grown in greenhouses. Furthermore, eggplants grown and harvested during the cool season were less susceptible than the summer crop. This has also been demonstrated for peppers and tomatoes. Even the time of day at which the crop is harvested can affect its storage characteristics (5). Of particular significance is the stage of ripeness of tomato fruit at the time of exposure to chilling temperatures. Unripe fruits are especially liable to low temperature damage, because the complex biochemical changes associated with the climacteric rise may be seriously disrupted, resulting in abnormal or arrested ripening (1,2). The severity of damage increases with duration of exposure.



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CONTROL Mature green tomatoes may be stored at 13° to 15°C for 1 or 2 weeks. 'Turning' tomatoes can be held at 10° to 12°C for a similar period. Ripe tomatoes can be stored at a lower temperature, provided that they can be utilised promptly on release from storage. Eggplants grown under hot conditions should be stored and transported at 10° to 12°C, while those grown in temperate countries or during the cool season may be kept at 8° to 10°C. Post-harvest 'conditioning' (1 day at 15°C and 1 day at 10°C prior to storage) permits storage at 7°C for several days without injury (3). Peppers should be held at 7° to 10°C, the appropriate temperature depending on provenance and season. Prevention of moisture loss helps to minimise the effects of chilling, so plastic film packaging is beneficial (4).

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See also:

EFIUVWEVWERE B.J.O. & THORNE S.N. (1988) Development of chilling injury symptoms in stored tomato fruit (*Lycopersicon esculentum* Mill.). *Journal of the Science of Food and Agriculture* **44**, 215–226 illus.

OTHER DISORDERS of tomatoes, peppers and eggplants

BLOTCHY RIPENING of tomatoes has also been called **cloud** (3). Another term, **graywall** (q.v.) was originally applied to this disorder but is now reserved for a condition induced by certain bacteria. Blotchy ripening occurs primarily in early and mid-season tomato crops, and large-fruited cultivars are particularly prone. The disorder is characterised by green, greenish-yellow or pale waxy areas on red fruit (cf. bronzing, q.v.). The affected part, which is usually near the calyx-end, remains firmer than the rest of the tissue which softens and appears to ripen normally. If an affected fruit is cut open it may be possible to discern a browning of vascular strands in the outer wall, together with the presence of abnormal firm white tissue (10). The causes are complex and are still being elucidated, but it seems that both nutrition and environment play a role. Numerous studies have demonstrated the importance of adequate potassium fertilisation if blotchy ripening is to be minimised (1,3,8). In protected crops it is advantageous to maintain the air temperature below 30°C (3).

BRONZING of tomatoes has also been referred to as **internal browning**, **vascular browning** and **brownwall** (5). It is usually the calyx-end of the fruit which is affected, and discoloration of internal tissue is often visible through the skin (3). If the fruit is cut open, brown corky tissue can be seen in the fruit wall and elsewhere, occasionally accompanied by white tissue. Symptoms of graywall (q.v.) are very similar. Affected fruits ripen to red and yellow, with no residual green colour (cf. blotchy ripening, q.v.). Bronzing is induced by the tobacco mosaic virus (TMV) attacking tomato plants at a particular stage in their development (5). Crops with rank or 'soft' growth are especially susceptible. Control measures include the use of TMV-resistant cultivars and, in susceptible varieties, treatment of seed to inactivate the virus (3).

CRACKING or **SPLITTING** of tomatoes takes several forms. Cracks at the calyx-end tend to be either radial or in concentric arcs. They are usually initiated before harvest and may heal, with the formation of unsightly scars. Worse, the cracks may become infected, later resulting in decay. This frequently happens when fruits split or burst open as a result of sudden uptake of water, either in the field or in dump tanks after harvest. Cracking and splitting are inherited tendencies, and cultivars differ greatly in susceptibility (3). Incidence of these disorders can be minimised by adequate calcium nutrition and, in dry weather, frequent irrigation to prevent drought stress. A separate disorder, characterized by very fine cracking, is known as russeting (q.v.).

GOLD FLECK and **GOLD SPECKLES** can affect the shoulders and calyx-end of tomatoes. Both types of disfigurement are associated with the formation of calcium oxalate crystals. Influential factors include cultivar, nutrition and greenhouse environment.

GRAYWALL of tomatoes has also been called **vascular browning**, and the symptoms are very similar to those of bronzing (q.v.). Greyish-brown areas can be seen through the skin, and the damaged parts ripen more slowly than the surrounding tissue (3,8). Graywall can be induced by several types of bacteria, including *Erwinia herbicola* (page 240) which is common in soil (8). Disease expression is increased by prior infection with tobacco mosaic virus and by exposure of the fruits to chilling temperatures, probably because tissue resistance is thereby lowered. Control measures include appropriate potassium fertilization (8) and protection from viral infection and chilling (3).

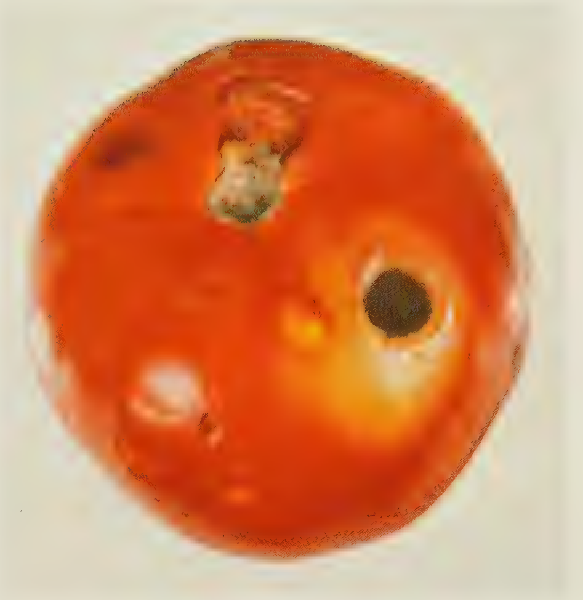
GREENBACK or **GREEN SHOULDER** of tomatoes is a condition in which the area around the calyx-end remains firm and green whilst the rest of the fruit ripens (3,6). The affected area may turn yellow (hence **yellowback** or **yellow shoulder**) but the line of demarcation between normal and disordered tissue remains distinct. Predisposing factors include exposure to excessive heat and an inadequate supply of potash and phosphate fertiliser. Susceptibility to the disorder is genetically determined and the gene for 'uniform ripening' has been incorporated into several cultivars (3). It should be noted, however, that some consumers prefer tomatoes with greenback symptoms.

GREEN PITTING or **BLACK SPOT** of bell peppers has caused serious losses through culling of blemished fruit. The pits become apparent as fruit colour changes from green to red; they are irregular in shape, approximately 2 to 6 mm across, slightly sunken, and pale green, dark green or black (2,7). Pitting is associated with with high levels of calcium in the fruit (cf. blossom-end rot, q.v.), and so an altered nutritional regime may be appropriate (2). Early harvesting is another strategy, but the long-term objective is to breed resistant cultivars (7).

HOLLOWNESS in tomatoes is also known as **boxiness** or **puffiness**. Hollow fruits lack some of the pulp which normally surrounds the seeds. Affected tomatoes tend to be 'slab-sided' and light in weight, and can be separated from normal fruits by flotation in water. Hollowness is primarily a disorder of early glasshouse crops produced under low light intensity. It can be minimised by appropriate mineral nutrition (3).

RUSSETING of tomatoes is a fine cracking of the skin, which mars appearance and reduces shelf-life of susceptible cultivars. The disorder has caused significant losses in greenhouse crops in the Netherlands.

SUNSCORCH or **SUNSCALD** can occur in both attached and detached fruit (9), and results in 'bleaching' of the affected area; the weakened tissue may later be colonised by moulds, alternaria and cladosporium rots being the most characteristic types of decay. Sunscorch is essentially a form of heat injury, but light is also involved; mature-green tomatoes and peppers are especially susceptible and suffer injury when the process of photosynthesis is disrupted by excessive heat (9). In some countries day-time field temperatures can reach 35° to 40°C and fruit temperatures even higher. If thermal stress continues after harvest, ripening is inhibited and colour development impaired (4). Depending on circumstances it may be advisable to provide shade for the plants or alternatively to choose a cultivar with bushy foliage to provide cover for the developing fruits. It may be necessary to control powdery mildew which would otherwise result in defoliation and exposure of fruits. Post-harvest handling practices should be directed towards prompt reduction of fruit temperature to below 25°C (4).



110 Sunscorch followed by cladosporium rot

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CHAPTER 3

MISCELLANEOUS FRUIT VEGETABLES

Considered in this brief chapter are two vegetables of which the edible part is botanically a type of fruit. They are otherwise unrelated.

Okra

Okra, *Hibiscus esculentus* L. (syn. *Abelmoschus esculentus* (L.) Moench), is a tropical vegetable crop which is grown from seed. Believed to have originated in Africa, it is also popular in India, the Far East, the warmer parts of Europe, the southern USA, South America and the Caribbean islands. Alternative names include **lady's fingers, bhendi, bhindi** and **gumbo** (10).

The plant, a herbaceous annual, belongs in the family Malvaceae, and its flowers give rise to elongated fruits (capsules), commonly called pods. These are harvested when young and tender; frequent (even daily) harvesting is desirable since, if allowed to mature, the pods become fibrous and tough. The seeds within the young fruit are surrounded by mucilage, which gives okra its characteristic culinary properties (10). Okra is more often eaten cooked than raw. In the USA a substantial proportion of the crop is grown for freezing, canning or preserving in brine (10).

Freshly harvested pods have a high rate of respiration and are prone to discoloration and toughening. Careful handling is essential, since bruising leads to blackening within a few hours. Lack of ventilation can result in self-heating of the pods and a fading of their green colour. Unless intended for immediate marketing or processing, pods should be cooled within a few hours of harvest. The use of iced water is inappropriate since it causes chilling injury, manifest as watersoaking and a reduced storage life (3,10). Air cooling is beneficial, the optimal conditions being a high humidity and a temperature between 7° and 10°C. At lower temperatures there is a risk of chilling injury, though trials have shown that susceptibility can be reduced by a post-harvest calcium dip or by storage in a modified atmosphere (4). In ordinary cool storage, healthy pods may be kept for 1 to 2 weeks before they become unpalatable as a result of toughening. Plastic film packaging is recommended as a protection against moisture loss and abrasion (3).

The majority of disease organisms affecting okra are seed-borne, and it may therefore be advisable to treat the seed with hot water before planting (8).

BACTERIAL BLIGHT, caused by *Pseudomonas syringae* pv. *syringae* (page 112) is reported from Romania and Brazil (5). It is essentially a field disease, with numerous small lesions first noticeable on leaves. Pods may also be affected, however; the reddish-brown spots are a disfigurement and may lead to significant rotting (5).

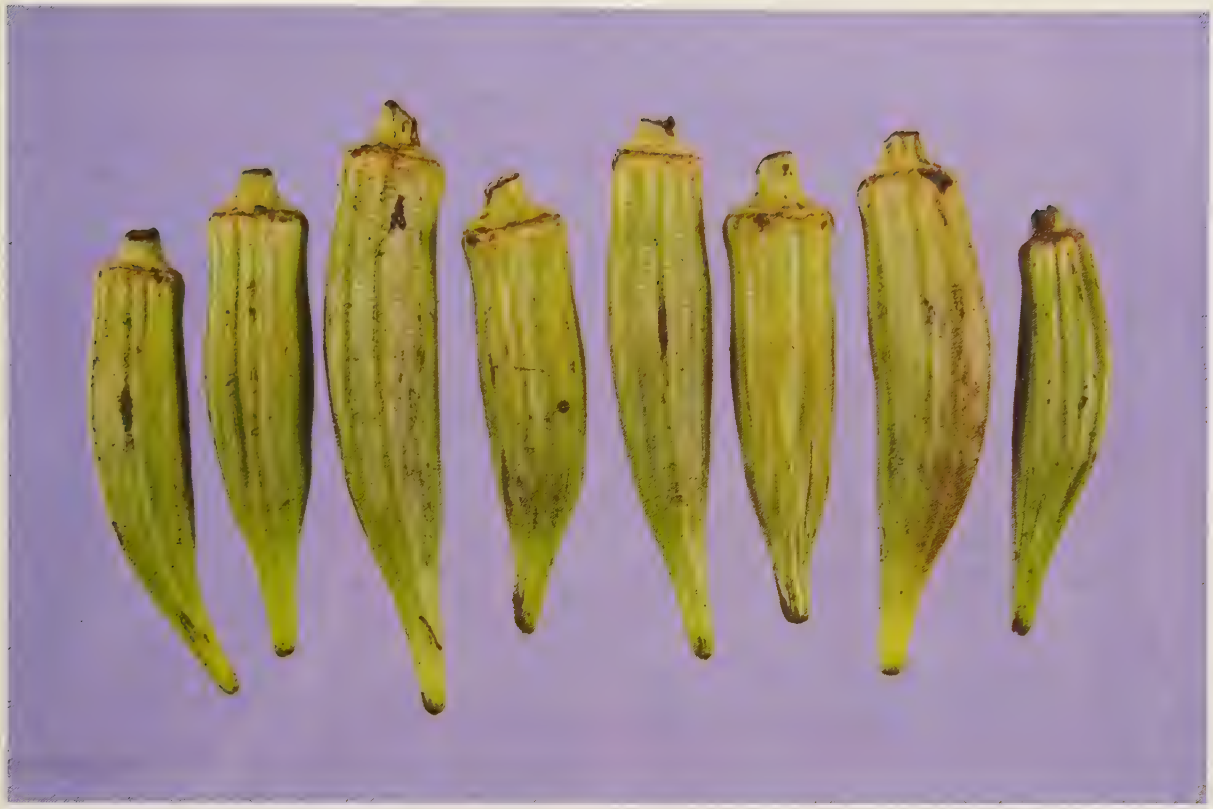
RHIZOCTONIA POD ROT, caused by *Rhizoctonia solani* (page 307), occurs in Nigeria, especially after wet weather (2). In Malaysia a species of *Rhizoctonia* was found to be capable of attacking young fruits whether or not they were wounded (8). The usual mode of transmission is through infected seed, which should therefore be treated before planting.

RHIZOPUS ROT, caused by *Rhizopus stolonifer* (page 81), has caused losses in Romania (7). Decayed tissue is brown, soft and sticky and, in a humid atmosphere, gives rise to coarse mould strands bearing characteristic spore heads. The fungus is reported to be seed-borne (2).

SOUR ROT, caused by *Geotrichum candidum* (page 288), has been recorded in markets in Egypt (1). Infected tissue is not discoloured but becomes soft and wet, with a sour odour. Complete decay can occur within a few days.

Other organisms causing pod rots of okra include *Fusarium solani* (2), *Phoma exigua* (6) and *Phytophthora palmivora* (2). *Choanephora cucurbitarum* is primarily known for causing young okra fruits to abort and fall from the plant (2). The same fungus can also infect pods at a later stage of their development (8).

WARTY POD is a physiological disorder which has caused losses in Japan (9). Nutritional factors appear to be instrumental, in particular a deficiency of nitrogen.



111 Senescent okra pods

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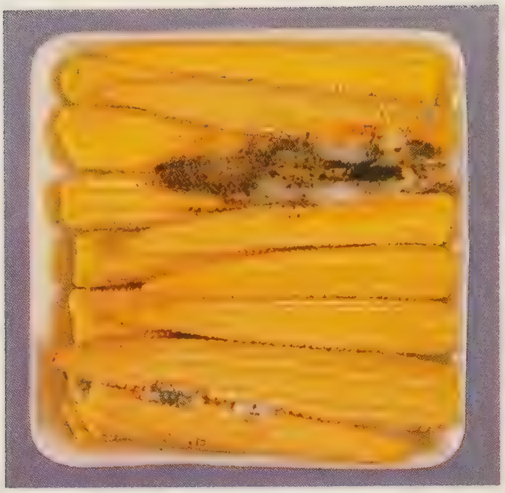
Sweet corn



112 Various moulds on 'baby corn'

Sweet corn, *Zea mays* L. var. *saccharata,* is a type of maize (6,9). Originating in Mexico, maize was one of the first plants to be domesticated, and remains one of the world's most important crops. It belongs in the grass family, Gramineae, and in America is known as corn (8). Male and female flowers are borne in separate inflorescences on the same plant. After fertilisation, the female florets give rise to grains (single-seeded fruits known as kernels in the USA and Canada) which are arranged in rows along the cob (ear). Most types of maize or corn have starchy grains, which are stripped from the cob, dried and used as a cereal. In the 18th century sugary types were described, and in recent years several 'sweet' and 'supersweet' cultivars have been bred for use as a succulent vegetable (2,3,6). The cobs are harvested when the kernels are pale yellow, plump and milky (4,9). Sweet corn can be marketed as separate kernels (often canned or frozen) or as 'corn-on-the-cob', a typical cob being 15 to 20 cm in length and still partially ensheathed in its green husk (7,9). 'Baby corn' consists of naked miniature cobs about 5 to 10 cm in length; this commodity (which is eaten whole) is currently grown in the Far East and air-freighted to Europe.

Post-harvest life is brief, since sweet corn respires at a high rate and sugar is rapidly converted into starch (2,4). At 25°C as much as half of the total sugar content is lost within 24 hours (10). Therefore, if deterioration in taste and texture is to be minimised, the product must be cooled immediately after harvest. Hydro-cooling is appropriate, and vacuum cooling is feasible if the cobs are pre-wetted (1). Top ice can be used to maintain a low temperature during transit (9). Corn maintained at 0°C loses sugar at only one sixth the rate shown by corn at 20°C, and can be held in satisfactory condition for up to a week. Plastic film packaging may be used, provided that the film is sufficiently permeable to preclude the development of off-odours (7). Modified atmosphere storage affords some benefit in that it retards sugar conversion and superficial mould growth. However, the main emphasis in research has been the breeding of especially sweet cultivars (10).

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CHAPTER 4
LEGUMES

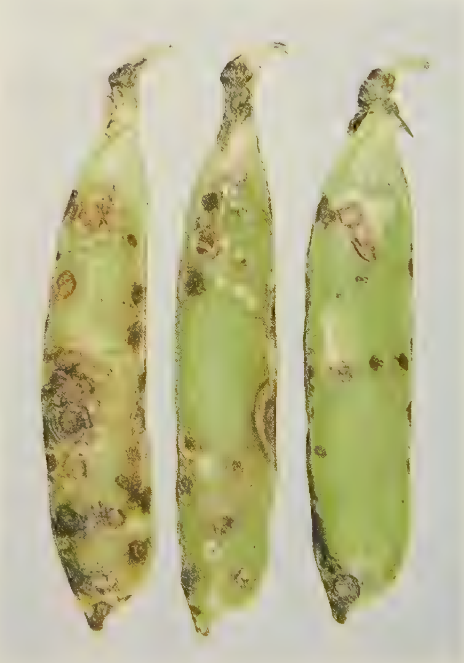
The family Leguminosae is of great importance in providing some of the world's most nutritious crops. As well as groundnuts (peanuts) and lentils, it includes peas and beans which exist in many diverse forms. All produce their seeds in fruit-pods known as legumes. These are rich in protein and, depending on cultivar, can be marketed fresh, sent for processing, or left on the plant to dry (pulses). The other significant property of leguminous plants is their ability to fix atmospheric nitrogen. They do this through beneficial bacteria which colonise their roots, with the added consequence that these crops can enrich the soil in which they are grown (4,5,6).

The **pea**, *Pisum sativum* L., originated in the Old World and has been cultivated since Neolithic times. Being a cool-weather crop, it is grown most extensively in temperate countries and in the highland tropics. In India peas are an important winter crop. World production of green peas (as distinct from dry peas) is nearly 5 million tonnes, the top ten producers being the USA, the UK, France, China, Hungary, the USSR, India, Italy, Belgium/Luxembourg and Romania.

Different pea cultivars are used in various ways. Edible-podded types have been bred with minimal 'parchment' (fibre) in the young pod-wall; they are known as sugar peas, snow peas or 'mangetout' (eat all) and are consumed in their entirety. Other cultivars have been bred specially for ease of mechanical harvesting and shelling for quick freezing, this industry being of considerable importance in Europe and North America (6). The entire crop of immature peas needs to be harvested within a matter of days, while all the peas are at a similar stage of maturity, before sugar turns to starch and before taste and texture deteriorate. For the same reason the harvested peas must be shelled and frozen within 2 or 3 hours of picking, or else immediately cooled pending treatment as soon as possible. Other methods of preserving peas include canning and hot-air dehydration. In some countries the success of the frozen pea industry means that nowadays a comparatively small percentage of pea production is marketed as fresh pods.

Whether in the form of flat snowpea-type mangetout, plump-podded sugar or 'jacket' peas, or as plump-podded fresh garden peas, harvested pods have an extremely high rate of respiration and consequently a very short shelf-life. Mangetout are often grown in the tropics and air-freighted to European markets where they command high prices as a luxury vegetable. Cool-chain distribution is necessary if quality is to be maintained, 0°C being optimal (3). Film-packaging serves to minimise moisture loss, but the film must be sufficiently permeable to preclude the possibility of anaerobic respiration and resultant off-odours (3). Fresh garden peas 'in shell' have declined in importance. They tend to be locally grown and are usually sold loose rather than packaged. They should be shelled and consumed immediately, before the peas become starchy and tough. If storage or long-distance transport is necessary, prompt refrigeration is essential; in the USA hydro-cooling has been successfully used, followed by 'top-icing' to maintain the temperature close to 0°C. These procedures have the added advantage of ensuring a high relative humidity which minimises moisture loss from the pods. Peas may be held for about 2 weeks under refrigeration, before they become undesirable in taste and texture.

The market quality of peas is reduced by the presence of blemishes on the pods. There are many causes, including bacterial blight and several types of ascochyta pod spot (4). If refrigeration is unavailable, pea pods may undergo self-heating and decay.

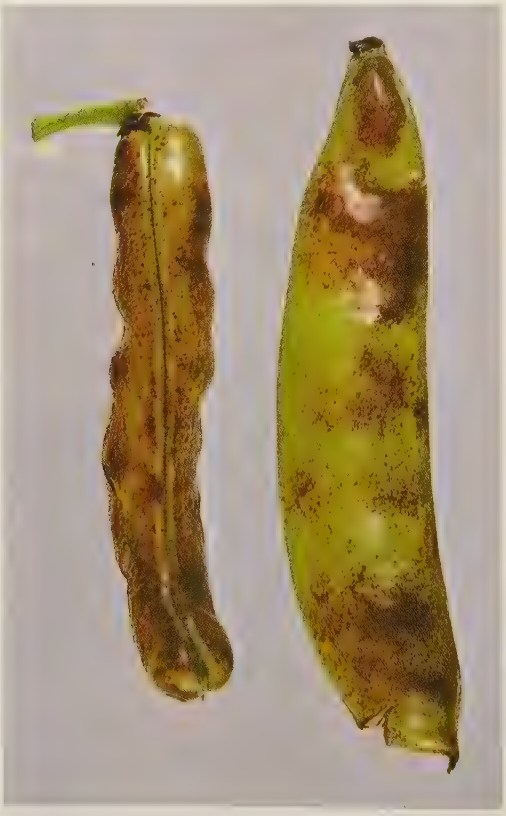


113 Mycosphaerella blight of peas

The **cowpea** or **blackeye pea**, *Vigna unguiculata* (L.) Walp., is thought to have originated in Ethiopia, and has for long been an important pulse crop in Africa and Asia (1). It reached the Americas in the 17th century. In the southern USA, where it is known as **southern pea**, the pods are harvested immature, mostly for freezing or canning. The shelled peas retain quality only for a matter of hours, and must be processed promptly. For southern peas in shell intended for the fresh market, pods should preferably be cooled to 4° or 5°C and marketed as soon as possible.

Throughout south and south-east Asia and in the West Indies, long-podded climbing varieties of *V. unguiculata* are widely used as a fresh vegetable, being known as **yardlong bean**. *V. radiata* (L.) Wilczek is the accepted name for **mung bean**, formerly classified in *Phaseolus*. Mung beans are especially important in India and China, where they are used in both the fresh and dry state. They are also utilized in the form of **bean sprouts**, that is the succulent shoots obtained within a few days of allowing the seeds to germinate in a warm moist environment. Harvested bean sprouts are highly perishable; they may be held for a few days at 0°C.

The **faba bean**, *Vicia faba* L., originated in the Near East and was cultivated by the ancient Egyptians, Hebrews, Greeks and Romans. It was the only edible bean known in Europe in pre-Columbian times, and was introduced into the New World after 1492.



114 Blemishes on faba beans

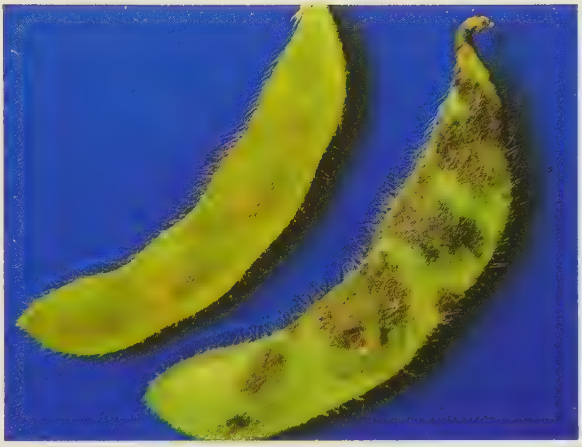
There are three kinds of faba bean, var. *minor* known as tick beans, var. *equina* known as horse beans or field beans (suitable for both animal feed and human consumption) and var. *major* known as broad beans. The last named is the main culinary type grown in northern Europe, where the seeds are marketed fresh, frozen or canned. In some Asian, African and Mediterranean countries faba bean seeds (harvested fresh or dry) provide a substantial part of the protein in human diet. China is by far the largest producer, followed by Ethiopia, Egypt, Italy and France. In Egypt faba beans are consumed daily, prepared in countless different ways (5). Not everyone derives nutritional benefit, however. Some individuals, genetically deficient in a certain enzyme, are predisposed to a blood-disorder (favism) induced by consumption of faba beans (5). Mediterranean peoples are peculiarly susceptible, and the victims tend to be male children. Symptoms of fatigue, pallor and jaundice develop within a few hours of ingestion, the active principle being vicine. Work is now in progress to breed cultivars low in vicine (5).

Fresh faba beans are highly perishable. Harvested pods intended for the fresh market should be despatched without delay, or else be promptly cooled to 0°C, at which temperature they may be held for about 2 weeks. Although the pods themselves are generally not consumed, their market value is reduced by the presence of blemishes or decay.

In contrast to faba bean, the genus *Phaseolus* is of New World origin, and has been cultivated in central and south America for several millennia. Five hundred years ago Spanish explorers took phaseolus beans across the Pacific to the Philippines and thence to the Asian mainland. They were also introduced into Africa, and reached Europe in the early 16th century. The **common bean**, *Phaseolus vulgaris* L., comprises an enormous diversity of market classes, including those known as French, kidney, navy and snap beans. *P. lunatus* L. is the **lima** or butter bean, while *P. coccineus* L. is the **runner bean**. Much effort has been devoted to the breeding of bean cultivars with resistance to the important field diseases, such as bacterial blights, angular leaf spot, anthracnose, white mould, ascochyta blight, rust, and (for lima bean) 'downy mildew' and phomopsis pod blight (q.v.) (1,7,10). Phaseolus beans are valued grain legumes or pulse crops in many tropical countries, whereas in temperate countries they are more usually grown for consumption in the green state. The main producers of green beans are, in order, China, Turkey, Italy, Spain, Egypt, the USA, Romania, France, Japan and Algeria, world production being approximately 3 million tonnes.

Phaseolus beans may be used as young pods (whole or sliced, fresh or frozen) and cultivars have been bred with reduced 'parchment' in the pod-wall and with minimal 'stringiness' of the sutures. Slender 'filet type' beans are classed as fine, very fine and extra fine. They are highly prized in Europe (especially in France), and there is a year-round trade in air-freighted fresh beans from African countries such as Kenya, Senegal, Mali, Burkina Faso and Egypt. Larger cultivars may be sliced diagonally, while yet others are shelled for use in soups and stews.

The harvested pods have a short storage life, little more than a week at 5° to 8°C. Lower temperatures induce chilling injury (q.v.), manifest on return to ambient temperature and characterised by surface pitting and russeting. On the other hand, higher-than-optimal temperatures result in premature senescence (yellowing, fibre-development) and decay, for example cottony leak, grey mould rot and watery soft rot (9,10). It is therefore important to ensure that pre-cooling is prompt and thorough, and that self-heating does not occur during transport and storage (8). A humid environment is essential for prevention of wilting; plastic film packaging is beneficial for this purpose and also for reducing mechanical damage during handling (2). Beans intended for processing may benefit from treatment with sulphur dioxide, which reduces discoloration of broken ends. Storage in a modified atmosphere may have potential for extending the holding time for beans awaiting processing.



115 Anthracnose of soybean

The **soybean**, *Glycine max* L., originated in China and has been grown in the Far East since early times. It was introduced into Europe in the 18th century and into north America in the 19th century, but it is only during the last 50 years that soybean has become of supreme importance in the USA. In America, however, the soybean is grown almost exclusively as a source of oil and meal, whereas in Asia it is eaten as a fresh vegetable as well as being processed in various ways. Like the mung bean (q.v.), it may be utilised in the form of **bean sprouts**.

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ALTERNARIA BLIGHT of peas and beans caused by *Alternaria alternata* (Fr.) Keissler

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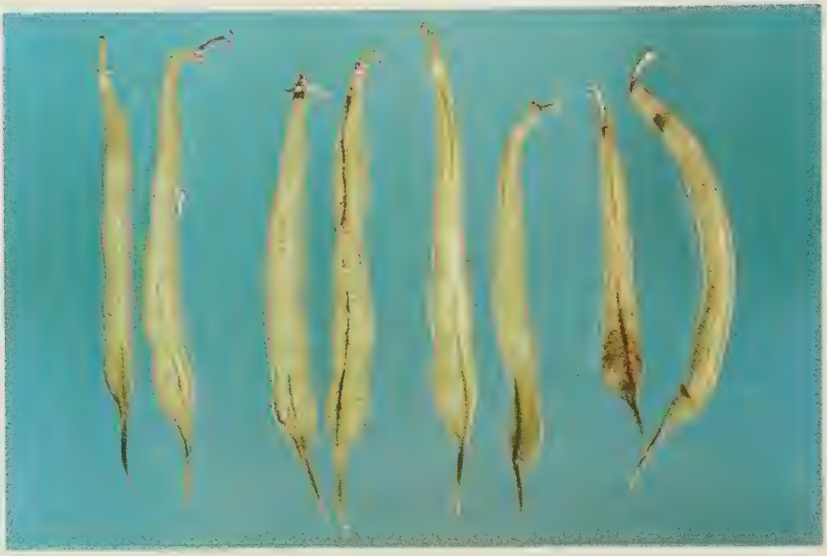

OCCURRENCE Alternaria blight has been recorded on peas in the USA (3,9) and India (8); on faba beans in Egypt and India (5); and on common beans in the UK, Germany, Romania (2), Canada (10), the USA (1) and Brazil (4,6). The commonest species is *A. alternata*, but *A. solani* (page 301) has been recorded on bean seeds in Zimbabwe (7).

SYMPTOMS On mature pea pods the symptoms are usually restricted to minute raised black flecks (9). On faba beans the circular to irregular black spots enlarge and coalesce, so that the entire pod becomes black (5). On common beans the first symptoms are small, irregular watersoaked flecks (1). Later these become reddish to dark brown or black, and may coalesce to produce long streaks. Under humid conditions there is a growth of black mould bearing characteristic chains of spores. Seeds may be discoloured, and this can be a serious problem in white beans (4,10); seed coats of the discoloured beans vary from light to dark grey and are occasionally streaked with brown (7). Affected bean seeds also tend to have wrinkled seed coats (7).

BIOLOGY The fungus survives on weed hosts (10) and on plant debris, and its spores (conidia) are dispersed by wind. Infection occurs only in wet conditions (4,9). Closely spaced or bushy plants are particularly susceptible because they retain drops of moisture (1). Some fungicides are ineffective against this fungus but, by successfully controlling other moulds, they predispose plants to alternaria blight. Infection of leaves leads to spotting, and sometimes shrivelling or defoliation (1,5,9). Infection of flowers is especially deleterious if the blossoms adhere to the pods (3). Pods may be attacked at all stages of development, and if wet weather prevails the seeds become stained (4,7,10). If the fungus penetrates the seed it tends to open the way for bacterial infection.

CONTROL During wet seasons field spraying is necessary, and the choice of fungicide is important. It is desirable to use cultural methods which result in well-aerated plants (1). Crop debris should be destroyed. Most pea cultivars appear to be susceptible (9), several faba bean cultivars are moderately resistant (5), and considerable variation is shown by common bean cultivars (1,10).

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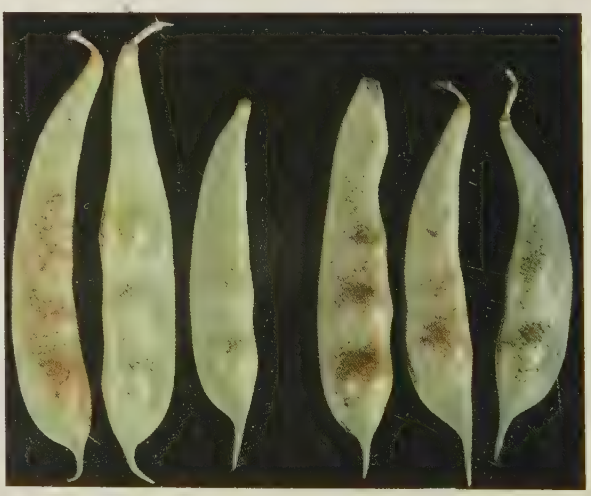
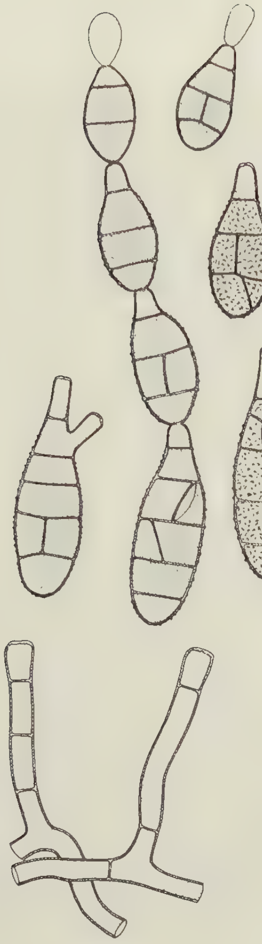
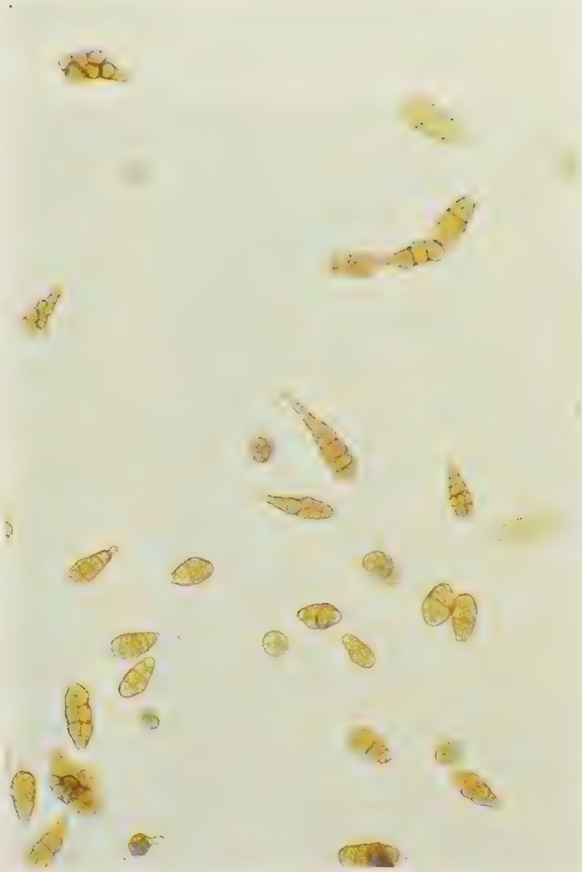




118 *Alternaria alternata* (CMI Drawing)
Conidia and conidiophores



119 Conidia of *Alternaria alternata*

1 ABAWI G.S., CROSIER D.C. & COBB A.C. (1977) Pod-flecking of snap beans caused by *Alternaria alternata. Plant Disease Reporter* **61**, 901–905 illus.

2 CRIŞAN A., BEDELEAN C. & IACOB R. (1979) [Aspects of the biology and control of *Alternaria* species on bean.] *Contribuţii Botanice 1979*, 241–250.

3 DILLARD H.R. & HUNTER J.E. (1988) Fungal-induced blemishes of snap peas associated with adherence of blossom parts to the pods. *HortScience* **23**, 847–848 illus.

4 GOMES J.L.L. & DHINGRA O.D. (1983) *Alternaria alternata* – a serious pathogen of white coloured snap bean (*Phaseolus vulgaris*) seeds. *Fitopatologia Brasileira* **8**, 173–177.

5 GURHA S.N., MISRA D.P., SHRIVASTAVA Y.C., SHUKLA A.K. & MISRA R. (1981) A new record of *Alternaria alternata* (Fr.) Keissler on *Vicia faba* Linn. and sources of resistance. *Indian Journal of Agricultural Sciences* **51**, 904–906 illus.

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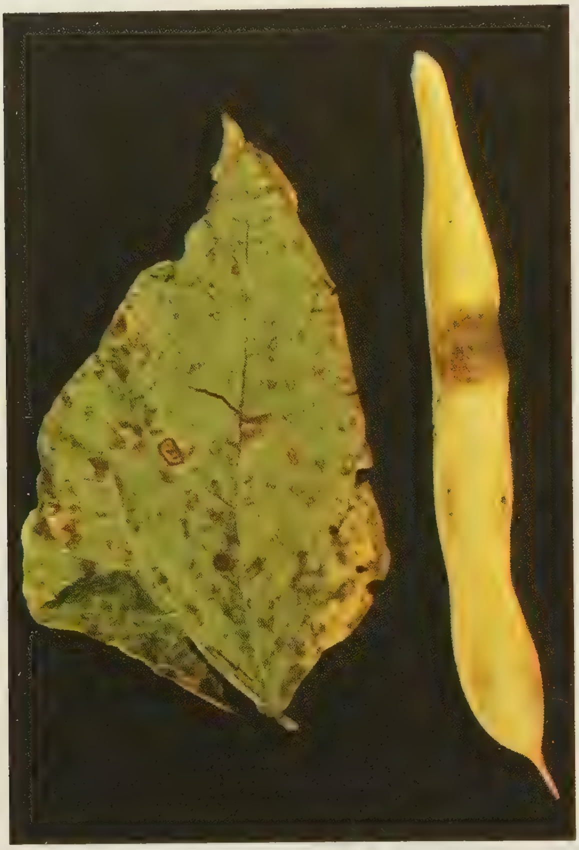
ANGULAR LEAF SPOT DISEASE of beans caused by
Phaeoisariopsis griseola (Sacc.) Ferr.

120



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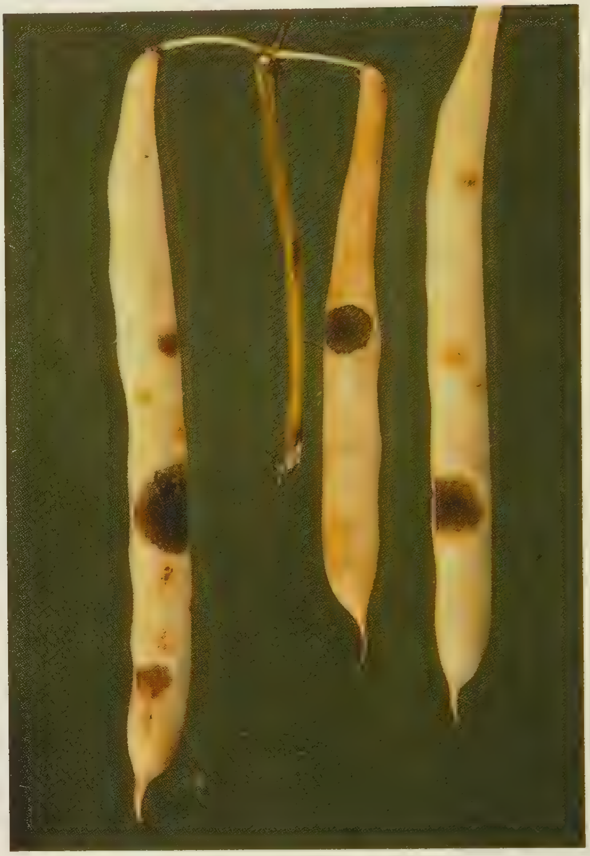
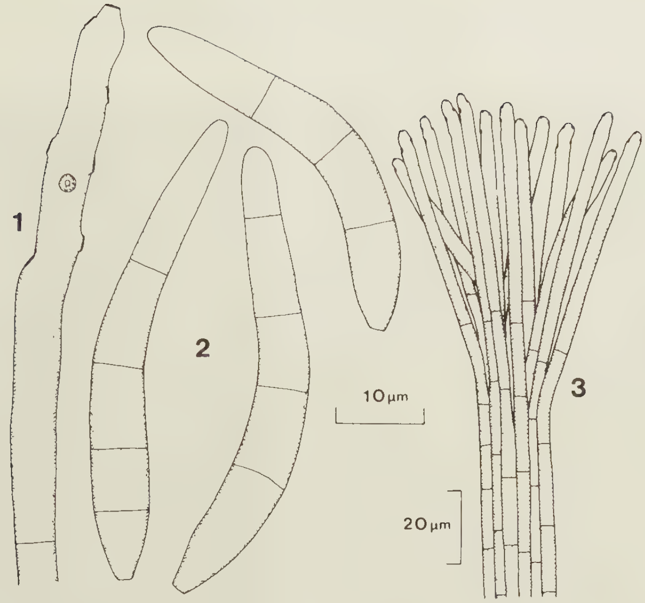


OCCURRENCE This disease of common and lima beans (2,3,8) has been recorded in most bean-producing areas (CMI Map 328) but appears to be of particular importance in the tropics and subtropics (3). Records include Argentina (8), Brazil (4), Colombia (1,3), Venezuela (5), India (9,10), Tanzania (6), the USA (3,7) and Australia (2).

SYMPTOMS Lesions on pods are roughly circular, and reddish brown with sharply defined borders, sometimes coalescing until much of the pod is affected (8). In humid conditions the lesions give rise to numerous minute black columnar structures (synnemata bearing conidia) (5,6).

BIOLOGY The fungus survives on infested crop debris, and in humid conditions produces asexual spores (conidia) which are dispersed by wind or water (3). In the presence of moisture the spores can cause infection of leaves via the natural openings (stomata), beneath which the fungus produces specialised thick-walled bodies (it is these which are later capable of surviving adverse conditions) (3,5). Leaf lesions, which are grey to brown, are typically angular in shape because their expansion is limited by the veins (1,3). In contrast, a particularly virulent form of this fungus (occurring in Tanzania) was found to produce circular lesions on leaves; furthermore the spore-bearing structures developed on both surfaces of the leaf, rather than only on the underside as in the common form (6). Spore development is favoured by periods of high humidity, whereas spore dispersal is at its most efficient in a dry atmosphere (3). Since, in turn, infection is favoured by cool, moist conditions, and disease development is most rapid during warm spells, epidemics occur only after alternating periods of different types of weather (7), perhaps aggravated by injudicious use of irrigation (1). Severe attacks result in defoliation (3,8). Even in mild outbreaks, lesions may form on stems and leaf stalks, and also on pods, which become unmarketable (10). In addition to substantial losses in yield (3), seed may be shrivelled (1) and of generally poor quality (5); furthermore, seeds directly beneath pod lesions may become infected (4). Although angular leaf spot is essentially a field disease, blemished pods may be encountered in the market.



122 *Phaeoisariopsis griseola* (from CMI Descr. No. 847, 1986)
1 conidiogenous cells 2 conidia 3 conidiophores in the form of a synnema

CONTROL Although it appears not to be very prevalent (5), seed transmission can be responsible for introducing the disease to new areas. Seed treatment may therefore be advisable. Cultural practices are of much greater significance in reducing the extent of disease. Crop debris should be destroyed or thoroughly ploughed in (5), and beans should be rotated with other crops (1). Good drainage is advantageous (5). Fungicide sprays may be used during the growing season (8,9). Different bean cultivars vary considerably in their susceptibility to angular leaf spot (2,5). Resistant cultivars are available (8), and breeding work continues; for example, crosses are made with the scarlet runner bean which is highly resistant (10).

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9 SINDHAN G.S. (1984) Evaluation of fungicides against occurrence of angular leaf spot of French bean caused by *Phaeoisariopsis griseola*. *Indian Journal of Plant Pathology* **2**, 39–42.

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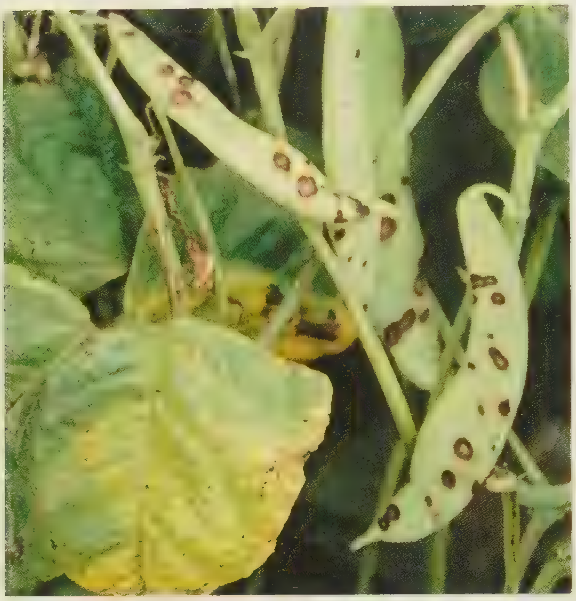
ANTHRACNOSE of peas and beans caused by *Colletotrichum* spp.

123



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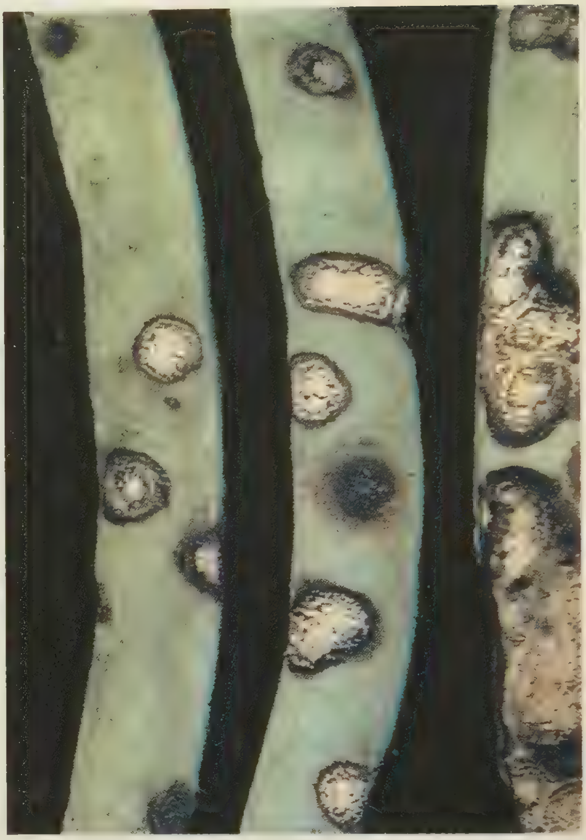
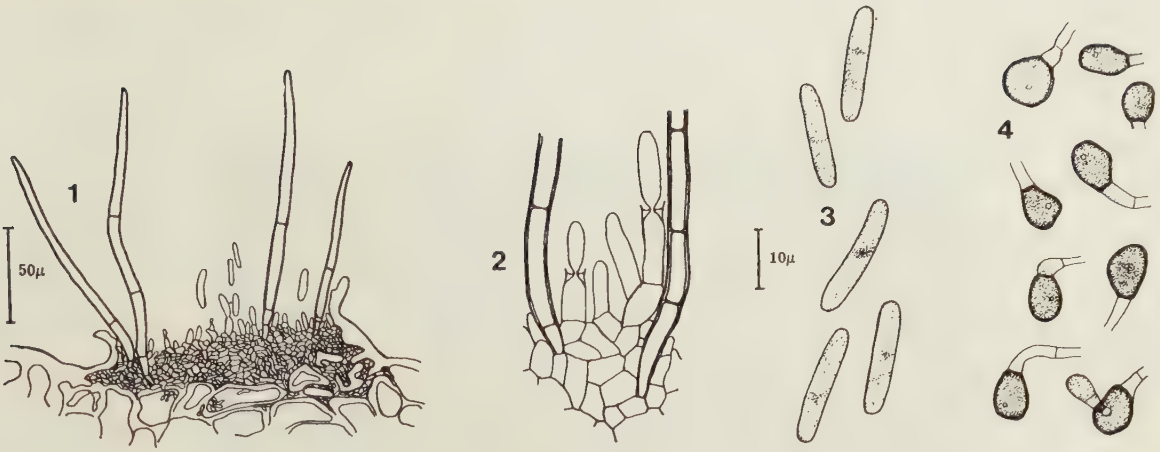


OCCURRENCE Bean anthracnose, caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Br. & Cav., occurs in all parts of the world where beans are grown (6). Records on common beans include Canada (9), the USA (1), Brazil (4), Colombia (8), Germany (5) and much of East Africa (6). This fungus has also been found to attack lima beans in the USA (1) and faba beans in Tanzania and China (10). *C. truncatum* (Schw.) Andrus & Moore occurs on lima beans (and occasionally common beans) in the USA (2). Pea anthracnose, caused by *C. pisi* Pat., is generally a minor disease, but losses have been reported from Japan, the USSR, Canada and the USA (3,7). It has been suggested that *C. pisi* is synonymous with *C. truncatum.* In parts of Europe the term 'anthracnose' is incorrectly used for ascochyta blight and pod spot (q.v.).

SYMPTOMS Reddish brown specks or blotches develop and may coalesce until much of the pod is discoloured (2,3). Individual lesions may become sunken, and are typically grey or black in the centre and bordered by a reddish brown margin (10). A humid atmosphere favours the production of characteristic salmon-coloured or flesh-coloured spore masses (conidia arising from acervuli), in which may sometimes be seen dark bristle-like projections (setae) (1,2,7). If dry conditions ensue, the spore masses tend to turn brown. *C. pisi* sometimes produces extremely small black bodies (microsclerotia) on the lesions (7). In severe anthracnose infections the seeds are invaded (1,9).

BIOLOGY Seed transmission occurs in faba bean (10), common bean (6) and lima bean (2); it appears to be of less importance in pea (3). The fungi are also able to survive from season to season in crop debris so long as it remains dry (3,9). The onset of wet conditions permits lesion development on seedlings grown from infected seed (1,9). Spores from these lesions and from infested debris are disseminated by watersplash, windblown rain, animals and man (1). Plants are susceptible to infection at all stages of growth, young tissue being especially vulnerable. The fungi produce swellings (appressoria) on the surface of leaves, stems and pods and, if moisture persists, are able to gain entry by direct penetration of intact tissue (2,5). In addition, wounds and lesions caused by other fungi predispose tissue to infection; for example, *C. pisi* may follow *Mycosphaerella pinodes* (the cause of mycosphaerella blight, q.v.) (7). During wet seasons crops may rot in the field (3); alternatively pods may appear healthy at harvest but undergo rotting during transit and storage (1,7). The optimal growth temperature lies between 20° and 25°C for *C. lindemuthianum* (1) and between 25° and 30°C for *C. pisi* and *C. truncatum* (2).



125 *Colletotrichum lindemuthianum* (from CMI Descr. No. 316, Mordue 1971) 1 acervulus 2 conidiophores 3 conidia 4 appressoria

CONTROL Crop rotation and field hygiene are very important; infested debris should be burned (1) or else ploughed in thoroughly to hasten destruction of the fungi (2,3,9). Clean seed is available from crops grown in semi-arid parts of the world (such as the western USA) where the disease is rare (9). Seed certification schemes are in force in some countries; fungicide treatment of seed with deep-seated infection is not always satisfactory. Protective sprays can be applied to the growing crop (2,4), but time of application is limited because of problems with residues. It is inadvisable to allow workers to move through the crop when rain water or dew is present on the plants (1). Despite the existence of several different races of these fungi (and the occasional development of new strains), considerable success has been achieved in the breeding of resistant cultivars (6,8,9,10).

---

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2 COX R.S. (1950) Stem anthracnose of lima beans. *Technical Bulletin of North Carolina Agricultural Experiment Station* No. 90, 28 pp illus.
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5 LANDES M. & HOFFMANN G.M. (1979) Zum Keimungs- und Infektionsverlauf bei *Colletotrichum lindemuthianum* auf *Phaseolus vulgaris*. *Phytopathologische Zeitschrift* **95**, 259–273 illus.
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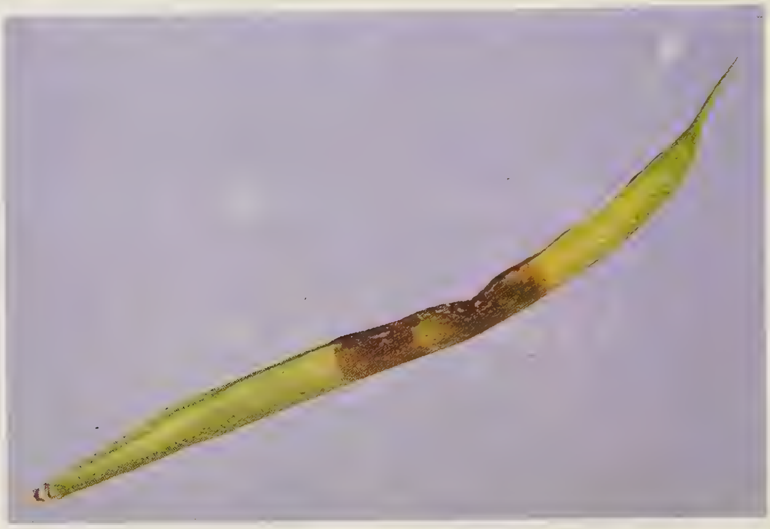
ASCOCHYTA POD SPOT of common beans and lima beans caused by

Ascochyta boltshauseri Sacc.
Ascochyta phaseolorum Sacc.

126



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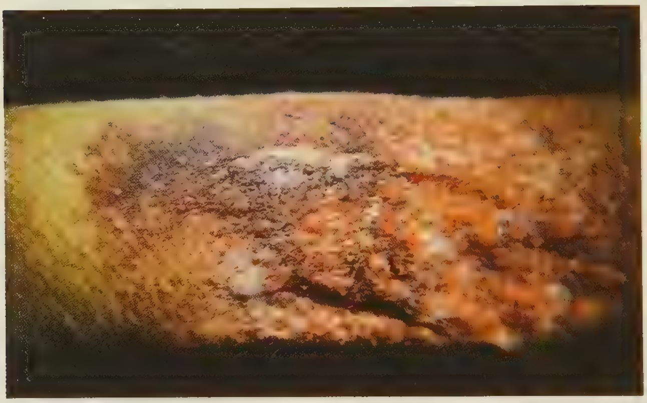
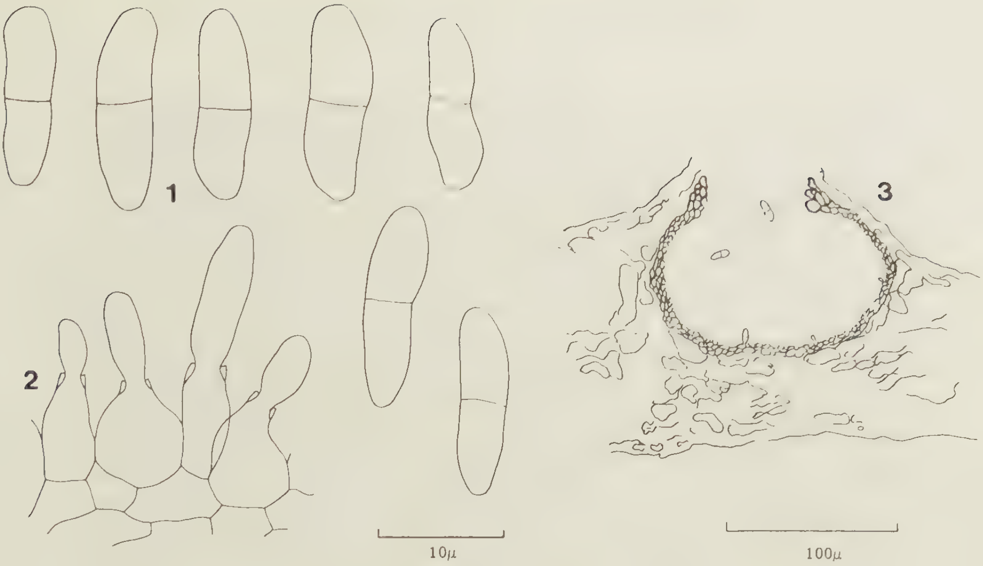


OCCURRENCE *Ascochyta boltshauseri* has been recorded on common beans in the USA (10), Germany (2,6) and the Netherlands (2,9). *A. phaseolorum* (which is synonymous with *Phoma exigua* var. *exigua*) (2) occurs on common beans in, for example, the USA (4), Colombia and Guatemala (8), Australia (1,7), New Zealand, Japan, the USSR (3), Germany (2) and the Netherlands (9). It has been recorded on lima beans in the USA (4), South Africa and Japan, on mung beans in Pakistan (5), and the author has observed it on filet-type beans imported into the UK from Kenya. *A. phaseolorum* may (3) or may not (9) be weakly parasitic on peas. A related fungus, *Phoma exigua* var. *diversispora* (Bubak) Boerema, is endemic in Western Europe and East Africa, and attacks common beans and cowpeas with great virulence (2). The following account is largely based on information relating to the two *Ascochyta* spp., but it is conceivable that the three fungi have sometimes been confused (2).

SYMPTOMS A dark dry stem-end rot may occur, accompanied by a darkening of the sutures along the length of the pod (7). Irregular reddish-brown lesions may develop elsewhere on the pod, sometimes in association with rust (q.v.) pustules (1). Minute brown bodies (pycnidia) develop on the lesions (1), often in concentric circles (6). Seeds may become infected, resulting in the formation of dark spots with greenish-grey edges (9).

BIOLOGY The fungi can survive in seed for at least two years, and probably also persist in infected crop debris (9). Both species exist in the asexual state (pycnidia giving rise to conidia) (3,4) and the spores are disseminated by watersplash (9). Infection generally occurs only during cool wet seasons (6) in crops which have been weakened in some way; plants become vulnerable after wind or insect damage or infection by other fungi (1). While leaves, stems and pods are attacked at injury sites and



128 *Ascochyta phaseolorum* (from CMI Descr. No. 81, Sutton & Waterston 1966) 1 conidia 2 conidiophores 3 pycnidium

primary fungal lesions, pods can also be infected via the moribund remains of the sepals (calyx), resulting in a stem-end rot (7). Decay continues after harvest, the optimal growth temperature for *A. phaseolorum* being approximately 25°C (4,5).

CONTROL Crop debris should be destroyed, and it is also important to control weeds, several of which can act as alternative hosts for these fungi (1,3). Clean seed should be obtained, since treatment of infected seed is not always satisfactory (6,9). Correct fertilisation and irrigation (coupled with protection from wind if necessary) help to ensure a healthy crop which can resist infection. Further protection can be given by fungicide sprays (6,7,8). It is preferable to harvest pods in dry weather and to cool them promptly, since decay is largely held in check at 10°C (4,5).

1 ALCORN J.L. (1968) Occurrence and host range of *Ascochyta phaseolorum* in Queensland. *Australian Journal of Biological Sciences* **21**, 1143–1151 illus.
2 BOEREMA G.H., CRÜGER G., GERLAGH M. & NIRENBERG H. (1981) *Phoma exigua* var. *diversispora* and related fungi on *Phaseolus* beans. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **88**, 597–607 illus.
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4 CROSSAN D.F. (1958) The relationships of seven species of *Ascochyta* occurring in North Carolina. *Phytopathology* **48**, 248–255 illus.
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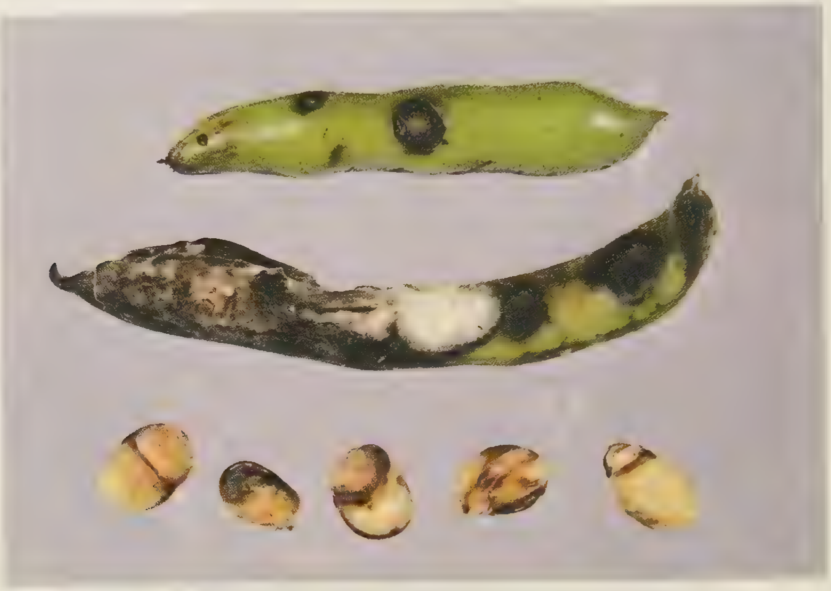
9 SNEEP J. (1945) De Ascochyta-vlekkenziekte van de boon (*Phaseolus*). *Tijdschrift over Plantenziekten* **51**, 1–16.
10 SPRAGUE R. (1935) *Ascochyta boltshauseri* on beans in Oregon. *Phytopathology* **25**, 416–420.

ASCOCHYTA POD SPOT of faba beans caused by ***Ascochyta fabae*** **Speg.**

129



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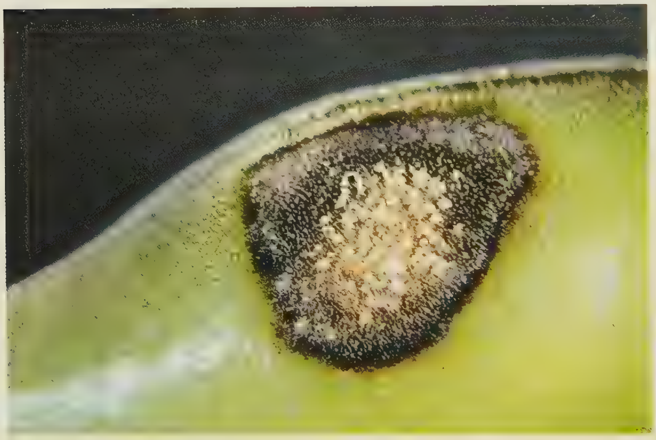
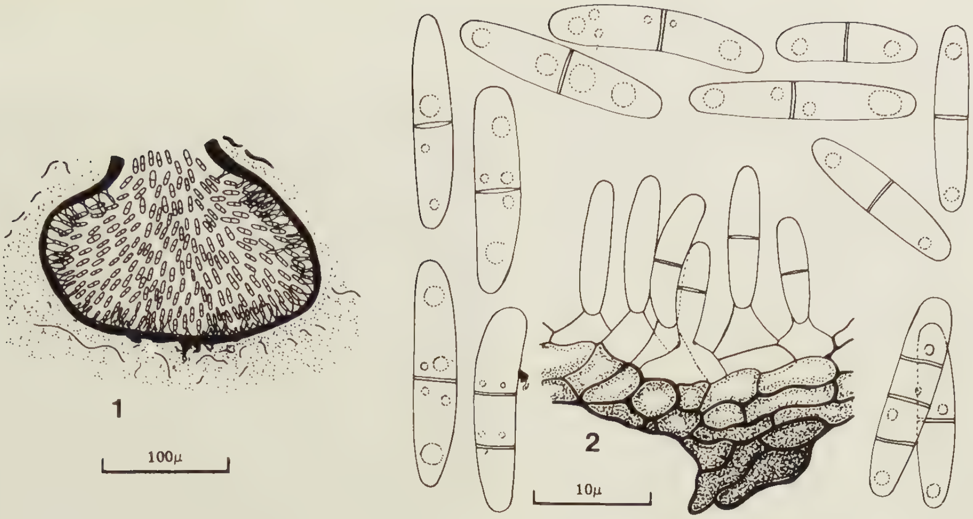


OCCURRENCE *Ascochyta fabae* occasionally causes heavy losses of faba beans (4). It has a wide distribution, including the UK (1,2,6), Denmark, Belgium (3), Iraq (8), Syria (5), the USSR, China (10), Canada (7,9), Australia and New Zealand (4). This fungus is largely confined to faba beans; it may (1) or may not (10) be weakly parasitic on peas. The pods of broad bean cultivars tend to be more vulnerable than those of horse beans and tick beans (6).

SYMPTOMS Spots on pods are circular to oval, often deeply sunken, and are dark brown edged with black (10). Lesions may coalesce until much of the pod is affected; furthermore, the seeds may be invaded (1). Minute brown bodies (pycnidia) form at the centres of the lesions or in concentric zones (10) and, in humid conditions, exude salmon-pink spores.

BIOLOGY The fungus survives in seed (4) and also in crop debris (8). Thick-walled resting spores (chlamydospores), resistant to freezing, have been found in crop residues in Canada. However, the fungus usually exists in the asexual state (pycnidia giving rise to conidia), either on dead stems and pods (3,10) or on volunteer bean plants outside the field (2). Wet weather induces exudation of spores, which are disseminated by watersplash (7), movement of personnel through the crop, and possibly wind-blown rain (3). Persistent moisture facilitates infection of leaves, stems and pods (1,10), the optimal temperature for disease development in the field being 20°C (9). Pods with incipient lesions are subject to post-harvest decay, the optimal growth temperature for the fungus being approximately 25°C (10).



131 *Ascochyta fabae* (from CMI Descr. No. 461, Punithalingam & Holliday 1975) 1 pycnidium 2 part of pycnidial wall, conidiogenous cells and conidia

CONTROL It is desirable to obtain clean seed (3), since disinfection treatments are costly (4) and not always effective (7,8). Seed certification schemes are in force in some countries but, in those with a wet climate, it is not always feasible to stipulate zero tolerance of ascochyta in the seed (2). The developing crop can be protected by fungicide sprays (3,4,7,8). Harvested pods should be kept cool, since the fungus grows scarcely at all at 10°C (10). There have been many recommendations about the importance of removal (8) or deep burial (3) of infected crop debris (10). In several localities, however, the role of 'escaped' bean plants is thought to be of equal (8) or greater (2) relevance to carryover of the fungus, which does not always survive long in crop residues (9). Thus appropriate control measures are dictated by local circumstances, climate, and season of the year (2). Work is in progress to breed faba beans with increased resistance to this disease (2,5,6); some cultivars tend to escape pod infection by virtue of their growth habit (6).

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1 BEAUMONT A. (1950) On the Ascochyta spot disease of broad beans. *Transactions of the British Mycological Society* **33**, 345–349 illus.

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3 DERENNE P. (1967) Ascochyta, parasite dommageable de la féverole. *Revue de l'Agriculture, Bruxelles* **20**, 845–849.

4 GAUNT R.E. & LIEW R.S.S. (1981) Control strategies for *Ascochyta fabae* in New Zealand field and broad bean crops. *Seed Science and Technology* **9**, 707–715.

5 HANOUNIK S.B. & ROBERTSON L.D. (1989) Resistance in *Vicia faba* germ plasm to blight caused by *Ascochyta fabae. Plant Disease* **73**, 202–205.

6 JELLIS G.J., LOCKWOOD G. & AUBURY R.G. (1985) Phenotypic influence on the incidence of infection by *Ascochyta fabae* in spring varieties of faba beans (*Vicia faba*). *Plant Pathology* **34**, 347–352.

7 KHARBANDA P.D. & BERNIER C.C. (1979) Effectiveness of seed and foliar application of fungicides to control *Ascochyta* blight of fababeans. *Canadian Journal of Plant Science* **59**, 661–666.

8 MICHAIL S.H., FARHAN M.A. & HUSAIN S.S. (1983) Sources of broad bean infection by *Ascochyta fabae* in Ninevah province of Iraq. *Seed Science and Technology* **11**, 393–402 illus.

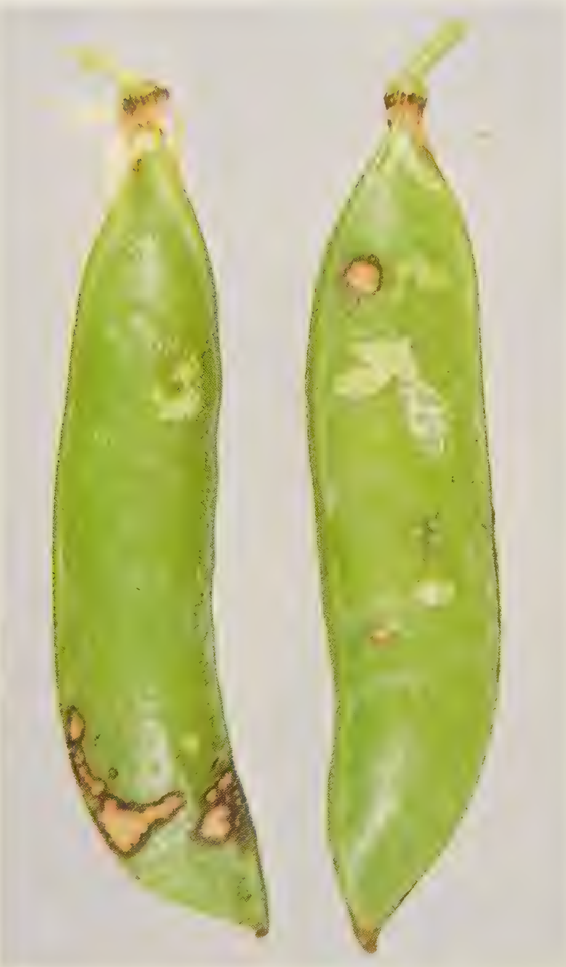
9 WALLEN V.R. & GALWAY D.A. (1977) Studies on the biology and control of *Ascochyta fabae* on faba bean. *Canadian Plant Disease Survey* **57**, 31–35.

10 YU T.F. (1947) Ascochyta blight and leaf and pod spot of broad bean in China. *Phytopathology* **37**, 207–214 illus.

ASCOCHYTA POD SPOT of peas caused by

Ascochyta pisi **Lib.**

132



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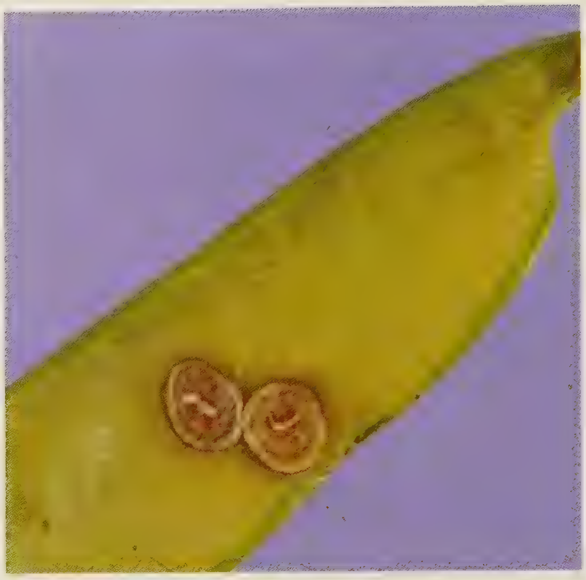


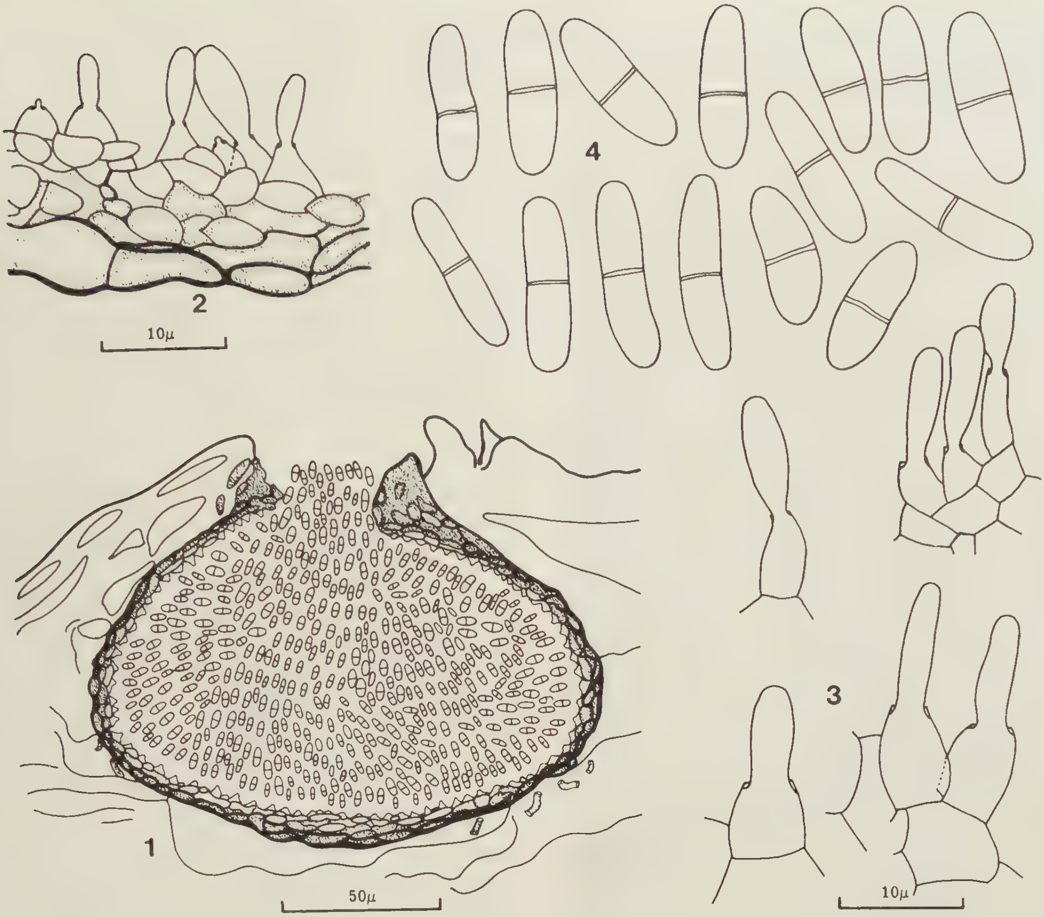
OCCURRENCE The fungus is widespread, occurring in most countries where peas are grown, for example the USA (5), Canada (9), Australia (1), India (7,8), Egypt (4), the UK (2,6) and the Netherlands (3). In parts of Europe the disease is loosely and incorrectly called 'anthracnose' (q.v.). Ascochyta blight may occur in conjunction with phoma rot (10) and/or mycosphaerella blight (q.v.) (1,4,5,7,9). *A. pisi* may also attack faba beans.

SYMPTOMS On fresh pea pods the spots are circular to irregular, and characteristically pale brown in the centre with a well-defined dark brown to black border (5). Later the lesions become deeply sunken, and small spherical bodies (pycnidia) develop which exude flesh-coloured 'tendrils' of spores. Moribund pods are subject to a generalised invasion, and pycnidia are produced throughout the tissue rather than within finite lesions (3).

BIOLOGY *A. pisi* appears to exist only in the asexual state (pycnidia giving rise to conidia) and, although it can survive for a limited period in crop debris (4,5), the most important means of transmission is in seed (3,4,6,9). Of plants grown from infected seed, some may die as seedlings, others develop stem, leaf and pod lesions, while yet others may be field-infected but remain symptomless. Disease can spread within a crop by means of spores produced on plant residues or on lesions of infected plants. Dissemination is by watersplash or wind-blown rain and, if moisture persists, the fungus can invade sound tissue; thus, in a wet season even low levels of initial seed infection can result in heavy losses (3). Incipient pod infections may be manifest as disfiguring blemishes by the time the crop is marketed.

CONTROL This disease has declined in importance since it has been effectively controlled by production of seed crops in dry areas (such as the western USA) where the fungus does not thrive (5,9). If healthy seed is not available by this strategy, seed may be disinfected by chemical treatment before sowing (3,6). If seed is stored, the level of infestation undergoes a natural decline. Pea cultivars differ in susceptibility to this disease but, because of the existence of different races of the fungus, a cultivar which shows resistance in one part of the world may not necessarily perform well elsewhere (1,2,7,8). Good field hygiene is desirable; crop debris should be removed and destroyed, or ploughed under to hasten destruction of the fungus (5).

Deutem



134 *Ascochyta pisi* (from CMI Descr. No. 334, Punithalingam & Holliday 1972) 1 pycnidium 2 part of pycnidial wall and conidiophores 3 conidiophores and conidia 4 conidia

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1 ALI S.M., NITSCHKE L.F., DUBE A.J., KRAUSE M.R. & CAMERON B. (1978) Selection of pea lines for resistance to pathotypes of *Ascochyta pinodes, A. pisi* and *Phoma medicaginis* var. *pinodella. Australian Journal of Agricultural Research* **29**, 841–849.

2 DARBY P., LEWIS B.G. & MATTHEWS P. (1986) Diversity of virulence within *Ascochyta pisi* and resistance in the genus *Pisum. Plant Pathology* **35**, 214–223 illus.

3 DEKKER J. (1957) Inwendige ontsmetting van door *Ascochyta pisi* aangetaste erwtezaden met de antibiotica rimocidine en pimaricine, benevens enkele aspecten van het parasitisme van deze schimmel. *Tijdschrifte Plantenziektenkundigedienst* **63**, 65–144 illus.

4 FADL F.A., NAGI I., EL-FANGARY I.M. & EL-SAWAH M.Y. (1985) Ascochyta diseases of peas and their control in Egypt. *Agricultural Research Review, Cairo* **63**(2), 127–136.

5 JONES L.K. (1927) Studies of the nature and control of blight, leaf and pod spot, and footrot of peas caused by species of *Ascochyta. Bulletin of New York Agricultural Experiment Station* No. 547, 46 pp illus.

6 MAUDE R.B., BAMBRIDGE J.M. & SPENCER A. (1986) Tests of fungicide seed treatments to eliminate seed-borne *Ascochyta pisi* (leaf and pod spot of peas). *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108**, Supplement), 70–71.

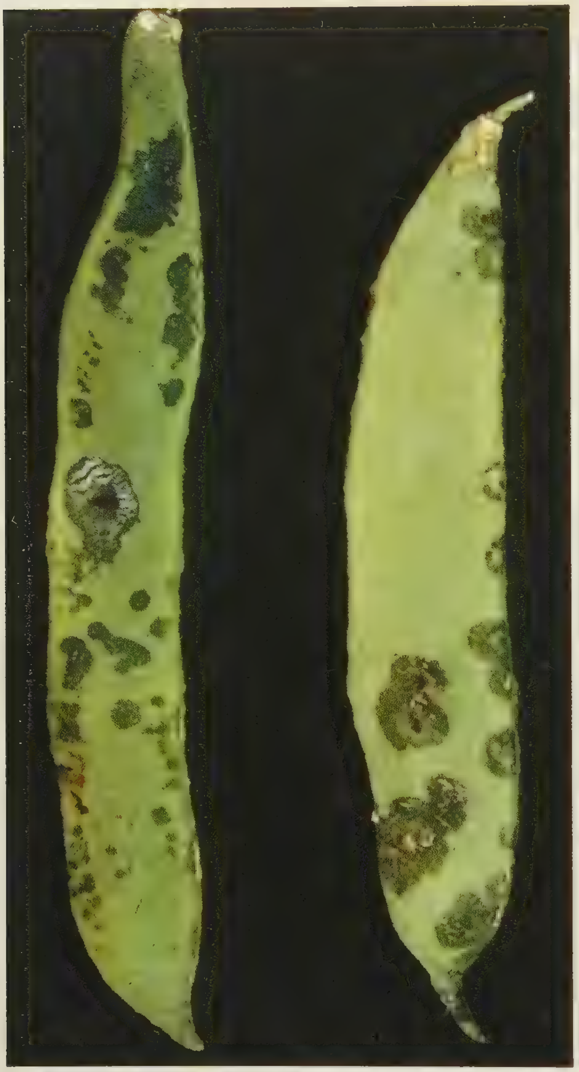
7 RASTOGI K.B. & SAINI S.S. (1984) Inheritance of seed shape and resistance to *Ascochyta* blight in pea (*Pisum sativum* L.). *Journal of Agricultural Science* **103**, 523–525.

8 SANDHU K.S. & DHILLON G.S. (1984) Reaction of pea varieties to *Ascochyta* blight. *Journal of Research of Punjab Agricultural University* **21**, 391–395.

9 SKOLKO A.J., GROVES J.W. & WALLEN V.R. (1954) Ascochyta diseases of peas in Canada – with special reference to seed transmission. *Canadian Journal of Agricultural Science* **34**, 417–428.

10 WHITE J.F. & MORGAN-JONES G. (1987) Studies in the genus *Phoma*. 6. Concerning *Phoma medicaginis* var. *pinodella. Mycotaxon* **28**, 241–248.

BACTERIAL BLIGHTS of peas and beans caused by
***Pseudomonas* spp.**
***Xanthomonas* spp.**



135 Halo blight of beans

OCCURRENCE *Pseudomonas syringae* pv. *phaseolicola* (Burkh.) Young et al. causes **halo blight**, which is of particular importance on common beans in temperate growing areas such as the UK (6), Italy (5), the eastern USA (8) and the cooler parts of South America. The same bacterium may also attack lima beans and, occasionally, peas (3).

Pseudomonas syringae pv. *pisi* (Sackett) Young et al. causes **bacterial blight of peas**, which has been recorded in, for example, the USA, South Africa (1), Australia (10), New Zealand, and parts of Europe. It is not considered to be fully established in the UK, and statutory action includes a ban on certifying seed from infected crops.

Pseudomonas syringae pv. *syringae* van Hall causes **brown spot blight** of common beans (9), lima beans (7) and peas (10), and the bacterium probably occurs in most areas where these crops are grown. A species of *Pseudomonas* has also been found on pods of faba bean (2).

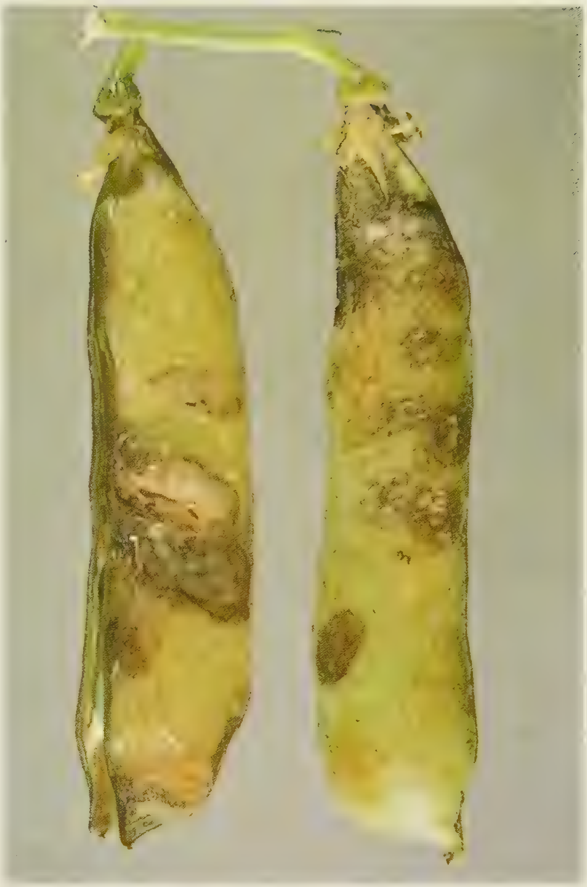
Xanthomonas campestris pv. *phaseoli* (Smith) Dye causes **common blight**, which is widespread but which tends to be most severe in the tropics and in warm temperate areas such as southern Europe and south-western USA. The bacterium attacks common beans (9), lima beans and, occasionally, peas.

Xanthomonas campestris pv. *phaseoli* var. *fuscans* causes **fuscous blight**, which is often found in association with common blight.

SYMPTOMS Watersoaked spots develop on the pods. Halo blight lesions tend to remain greyish-green and there may be a pale exudate; the disease is sometimes called **greasy spot** (the 'halo' symptom being confined to leaf lesions). Common and fuscous blight lesions on beans are often zonate, with narrow, reddish-brown borders, and any exudate is yellowish. Brown spot blight is characterised b lesions which remain very small and turn reddish-brown, while in bacterial blight of peas the water-soaked spots are irregularly shaped and slightly sunken. Seeds directly beneath pod lesions are likely to be infected (6), but infected seed may also result from systemically infected plants with no obvious lesions. Symptoms are not always characteristic, and bacteriological tests are necessary to confirm identification.

BIOLOGY The bacteria may survive on weed hosts, in crop debris (1) and also in or on the seed (6,9). Infected seeds may give rise to plants which are infected throughout (systemic infection resulting from bacteria transported through the vascular system). Local infection can occur from bacteria on the seed or on debris in wet soil. External lesions develop, and in humid conditions bacteria are exuded. Dissemination is by irrigation water (9), wind-blown rain (8) or wind-blown sand (1), and new infections occur via the plant's natural openings (stomata) or through wounds. Frost-injured pea plants are especially susceptible to bacterial blight (1). Optimal conditions for disease development differ, for example the optimal field temperatures for halo blight and common blight are 16° to 20°C and 25° to 30°C respectively. Infected pods, though symptomless at harvest, may undergo serious rotting during transit and storage.

CONTROL Crop rotation is desirable, and correct choice of planting dates is important if the risk of frost damage is to be minimised (1). The use of low-lying land should be avoided for the same reason. Since a very small number of infected seeds can be sufficient to permit an epidemic (6,8), it is essential to use certified seed, which is usually produced in arid areas with ground-level irrigation (8). (Sprinkle irrigation favours development of these diseases) (9). Strict quarantine measures are necessary to keep the producing areas free from contamination by these organisms (9). There have been attempts to use bactericidal sprays on seedlings and at flowering time (5). Wind-breaks may be advisable (1). Some resistant pea and bean cultivars are available, and breeding work continues (4), complicated by the fact that some cultivars show resistance in temperate zones and yet are susceptible in the tropics (9).



136 Bacterial blight of peas

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1 BOELEMA B.H. (1972) Bacterial blight (*Pseudomonas pisi* Sackett) of peas in South Africa, with special reference to frost as a predisposing factor. *Mededelingen Landbouwhogeschool Wageningen* **72**(13), 87 pp illus.
2 GRIESBACH E. (1976) Die Bakteriosen der Ackerbohne – Symptombild und Erreger. *Nachrichtenblatt für den Pflanzenschutz in der DDR* **30**, 233–236 illus.
3 GUTHRIE J.W., HUBER D.M. & FENWICK H.S. (1965) Bacterial blight of peas incited by *Pseudomonas phaseolicola* (Burk.) Dows. *Plant Disease Reporter* **49**, 854.
4 SCHUSTER M.L. & COYNE D.P. (1981) Biology, epidemiology, genetics and breeding for resistance to bacterial pathogens of *Phaseolus vulgaris* L. *Horticultural Reviews* **3**, 28–58.
5 TAMIETTI G. (1984) Tentativi di lotta chimica in campo contro *Pseudomonas phaseolicola* (Burkh.) Dowson. *Informatore Fitopatologico* **34**(11), 27–30 illus.
6 TAYLOR J.D., DUDLEY C.L. & PRESLY L. (1979) Studies of halo-blight seed infection and disease transmission in dwarf beans. *Annals of Applied Biology* **93**, 267–277.
7 TISDALE W.B. & WILLIAMSON M.M. (1923) Bacterial spot of lima bean. *Journal of Agricultural Research* **25**, 141–153 illus.
8 WALKER J.C. & PATEL P.N. (1964) Splash dispersal and wind as factors in epidemiology of halo blight of beans. *Phytopathology* **54**, 140–141.
9 WEBSTER D.M., ATKIN J.D. & CROSS J.E. (1983) Bacterial blights of snap beans and their control. *Plant Disease* **67**, 935–940 illus.
10 WIMALAJEEWA D.L.S. & NANCARROW R.J. (1984) Bacterial blights of peas in Victoria. *Australian Journal of Experimental Agriculture and Animal Husbandry* **24**, 450–452.

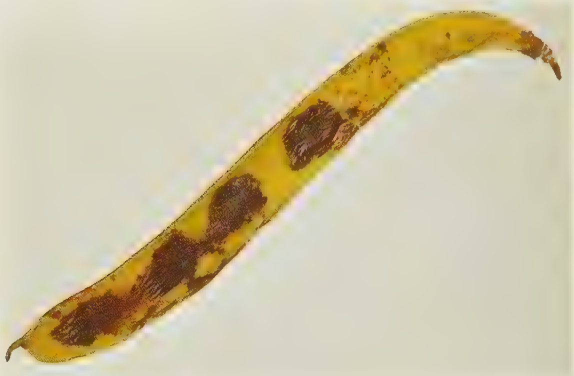
See also:

SCHWARTZ H.F. & McMILLAN M.S. (1989) Assaying bactericide efficiency against pseudomonads on bean leaf surfaces. *Plant Disease* **73**, 822–824.

CHOCOLATE SPOT AND GREY MOULD ROT of peas and beans caused by

Botrytis fabae **Sardiña**
Botryotinia fuckeliana **(de Bary) Whetzel**
Conidial state: *Botrytis cinerea* Pers.

137



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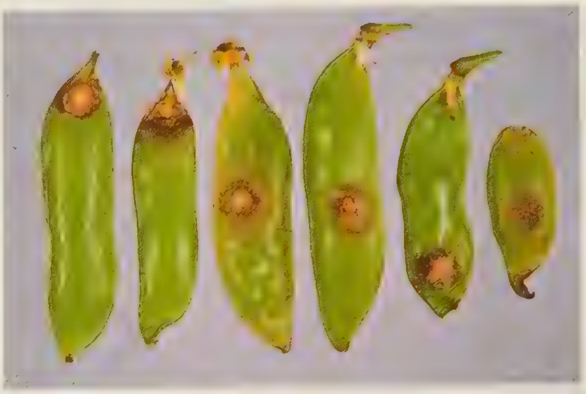
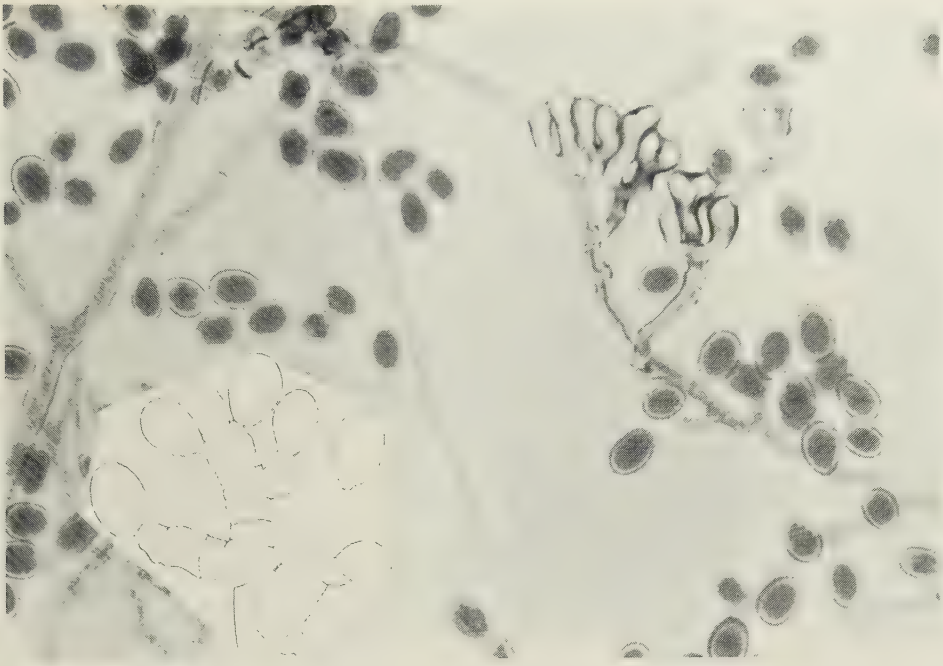


OCCURRENCE Chocolate spot and grey mould rot of faba beans may be caused by either *Botrytis cinerea* (5), *B. fabae* (1) or both (3,4,9). Records include Australia, South Africa, Egypt, Canada, the UK (1,3,5), Norway (9), Spain and Argentina (4). *B. cinerea* has been recorded on peas in, for example, France, Denmark, the Netherlands, the USA (2) and India (8); on common beans in the USA (7) and Belgium (10) and on lima beans in the USA (6). *B. fabae* occurs infrequently on these hosts.

SYMPTOMS In peas, common beans and lima beans brownish spots develop on any part of the pod (6,8). In faba beans there are reports of lesions being predominantly at the distal end (5) or along one side or edge of the pod (4); they either remain as non–progressive 'chocolate spots' or else coalesce and turn black (5). A humid atmosphere permits development of a greyish mould with grey–brown spores (conidia) (pages 151, 281) (6,8). Measurement of spore size serves to distinguish the two species (3). Eventually black resting bodies (sclerotia) (9) may form on the pod (4). In severe infections the seeds are invaded (5,8,9) and, in pea, take on a chalky texture.

BIOLOGY Besides surviving for a limited period in seed, these fungi persist as sclerotia on crop debris in the soil (1,9). *B. cinerea* may produce the sexual state (apothecia giving rise to ascospores) (3,7). Both fungi produce the asexual state (conidia) and spore dispersal is by air currents or watersplash (4,6). If moisture persists, spores can cause direct infection of leaves and stems of faba bean, especially after frost damage (1,3,9). In favourable conditions, restricted 'chocolate spot' lesions may suddenly enter the aggressive phase, characterised by rapid enlargement, darkening and copious spore production (1). Pods may also be attacked (4,9). *B. fabae* can be especially virulent (3,9). The characteristic mode of infection by *B. cinerea* is via moribund material, in particular the dying flowers of faba bean (5), kidney bean (10), lima bean (6) and pea (2). Infection may occur through flower parts still attached to the pod (5) or via cast blossoms lodging on any part of the plant (2,6). Cool wet conditions permit epidemic development of lesions on leaves, stems, flowers and pods (7). In peas, grey mould is reported to be especially severe after attacks of rust (q.v.) or powdery mildew (q.v.) (8). Pea and bean pods with incipient infection at harvest deteriorate during transport and storage (2), the optimal growth temperature for both species being approximately 20°C (7).

CONTROL Although infected seed has occasionally been responsible for the introduction of *B. fabae* to new areas, it is not the principal source of disease. More important than seed treatment are cultural practices designed to reduce crop susceptibility to infection and epidemic spread. Such measures (which are effective against both fungi) include correct irrigation and fertilisation, wide spacing and suitable orientation of rows to permit effective natural aeration so that drops of moisture evaporate promptly (6). If spots appear on the lower leaves before or during flowering, then a fungicide spray can be used to prevent epidemic development of *B. fabae* (1). Control of *B. cinerea* may require two sprays, the first applied at the onset of flowering



139 *Botrytis fabae* (from CMI Descr. No. 432, Ellis & Waller 1974) Conidia and conidiophores × 500

(10). In this species there are problems with the development of fungal strains tolerant of the usual fungicides (3). Consequently there is interest in biological control strategies, in particular spraying the blossoms with a suspension of a fungus which is antagonistic to grey mould but otherwise harmless. After harvest, infected crop debris should be destroyed or buried (incorporation in soil hastens natural decay of the sclerotia). Some faba bean cultivars have partial resistance to chocolate spot disease.

1 CREIGHTON N.F., BAINBRIDGE A. & FITT B.D.L. (1985) Epidemiology and control of chocolate spot (*Botrytis fabae*) on winter field beans (*Vicia faba*). *Crop Protection* **4**, 235–243.

2 FORD R.E. & HAGLUND W.A. (1963) *Botrytis cinerea* blight of peas associated with senescent blossoms in northwestern Washington. *Plant Disease Reporter* **47**, 483–485 illus.

3 HARRISON J.G. (1988) The biology of *Botrytis* spp. on *Vicia* beans and chocolate spot disease – a review. *Plant Pathology* **37**, 168–201.

4 JAUCH C. (1947) La "mancha chocolate" de las habas. *Revista de Investigaciones Agrícolas* **1**, 65–80 illus.

5 JELLIS G.J. & BOND D.A. (1980) Observations on the incidence of *Botrytis* pod rot on *Vicia faba*. *Plant Pathology* **29**, 66–69 illus.

6 KENDRICK J.B. & MIDDLETON J.T. (1950) Gray mold of lima beans. *Phytopathology* **40**, 228–234 illus.

7 POLACH F.J. & ABAWI G.S (1975) The occurrence and biology of *Botryotinia fuckeliana* on beans in New York. *Phytopathology* **65**, 657–660 illus.

8 SINGH U.P., SINGH H.B. & SINGH R.B. (1982) *Botrytis cinerea* causing grey mould of pea. *FAO Plant Protection Bulletin* **30**, 159–160 illus.

9 SUNDHEIM L. (1973) *Botrytis fabae, Botrytis cinerea* and *Ascochyta fabae* on broad bean (*Vicia faba*) in Norway. *Acta Agriculturae Scandinavica* **23**, 43–51 illus.

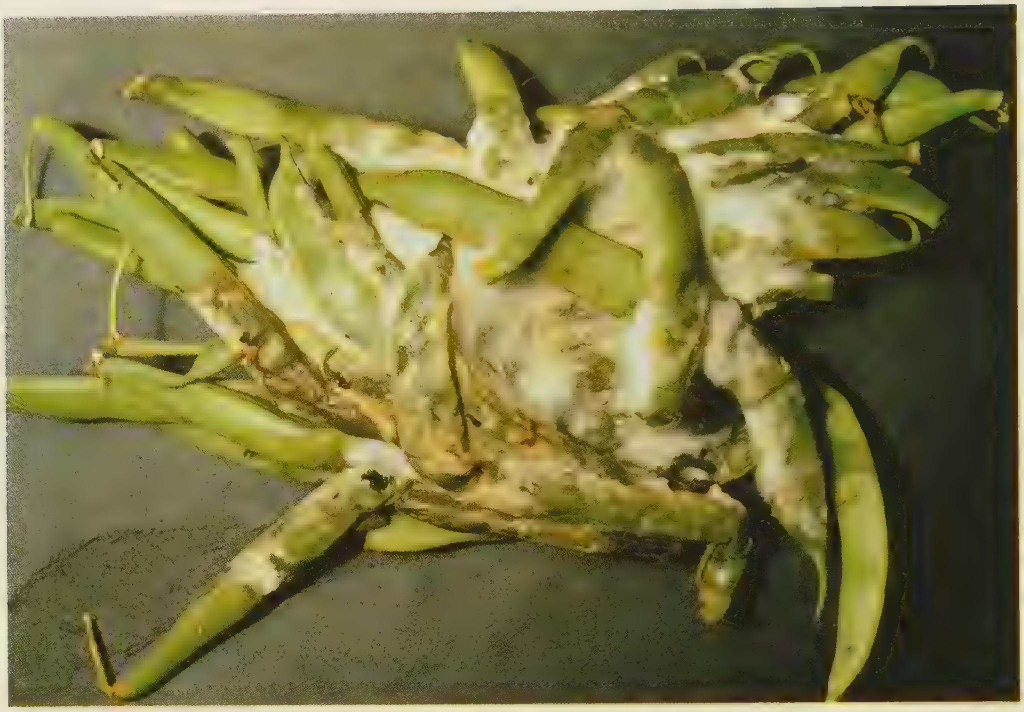
10 VULSTEKE G. & MEEUS P. (1982) Chemical control of *Botrytis cinerea* and *Sclerotinia sclerotiorum* on dwarf snap beans. *Netherlands Journal of Plant Pathology* **88**, 79–85.

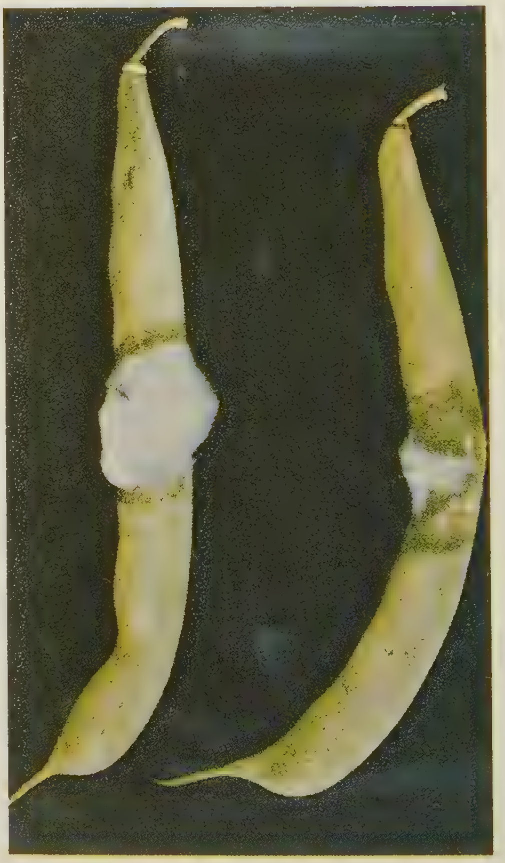
See also:

NELSON M.E. & POWELSON M.L. (1988) Biological control of gray mold of snap beans by *Trichoderma hamatum*. *Plant Disease* **72**, 727–729.

COTTONY LEAK of beans caused by *Pythium* spp.

140



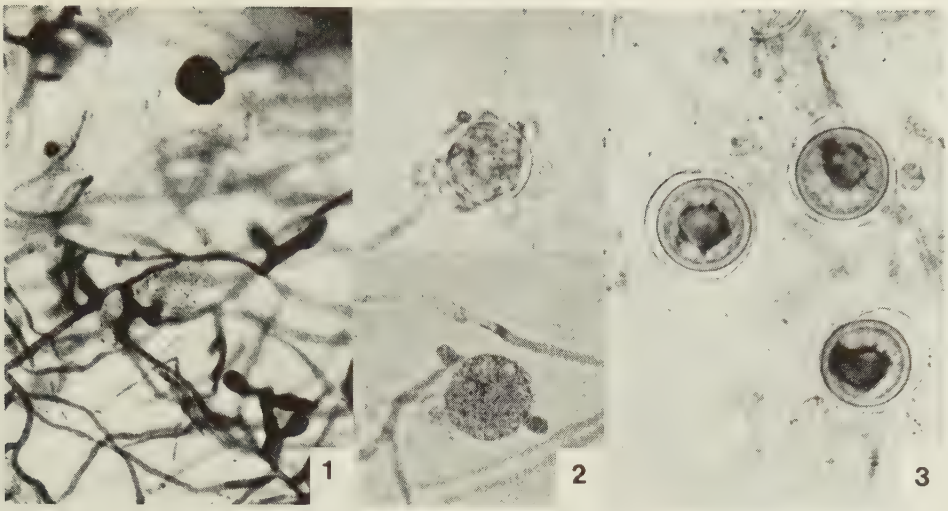


141

OCCURRENCE Pod rot and cottony leak of common beans may be caused by *Pythium aphanidermatum* (4,6,8), *P. butleri* (10) or *P. debaryanum* (1,2). Other species found on common beans include *P. myriotylum* (5) and *P. ultimum* (1). *P. ultimum* may also be associated with lima bean and pea plants (3,7), while *P. graminicola* has been recorded on faba bean (9). Most records of pythium pod rot are from the USA (1,4,10) and India (2,8,9).

SYMPTOMS Initially pods may be disfigured by dark lesions of irregular shape which enlarge rapidly in warm conditions (8,9). In a humid atmosphere there is copious development of white cottony mould (1), the strands being more delicate than those associated with sclerotinia rot (watery soft rot, q.v.). Infected pods are bound together by wefts of mould, and liquid may leak from the rotting tissue (2,4).

BIOLOGY These fungi survive in the soil in the sexual state (thick-walled oospores) or the asexual state (sporangia which may give rise to zoospores); furthermore, mould strands can persist in crop residues (5). In wet conditions spores or mould fragments are splashed on to the aerial parts of plants and, if moisture persists, infection of stems, leaves, flowers or pods can occur (1,3,6,8,9). Penetration may be direct or via natural openings



142 *Pythium myriotylum* (from CMI Descr. No. 118, Waterhouse & Waterston 1966) 1 sporangia × 300 2 young oogonia with antheridia × 500 3 mature oogonia with oospores × 500 (stained with cotton blue in lactophenol)

(stomata); the presence of injuries predisposes plants to infection (1). Disease development is most rapid at high temperatures (25° to 30°C) and high humidity (such as is produced by overhead sprinklers) and there may be severe losses in the field (1). Infected pods are reduced in value by disfiguring lesions (8,9) or, if stored in a warm environment, may undergo serious rotting. The moulds can spread into adjacent healthy pods, resulting in 'nests' of decay (4).

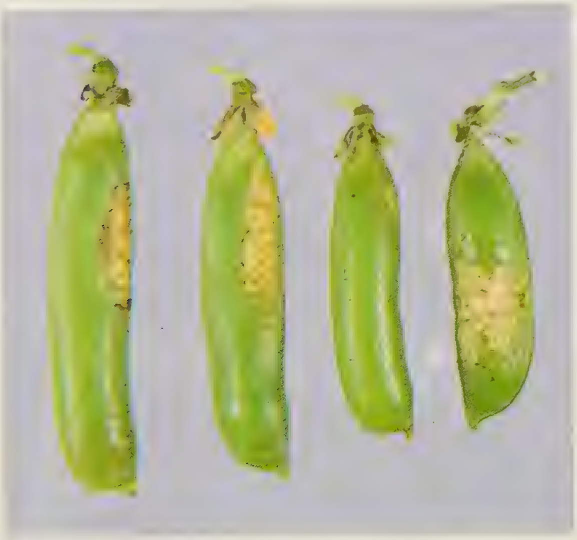
CONTROL Crop debris should be promptly removed and destroyed, so as to minimise sources of infection. Seed may be treated with fungicide to protect it against attack from fungi remaining in the soil (7). Excessive irrigation of developing plants should be avoided. Pods should be harvested in dry weather and graded with care (2,4). Post–harvest treatments are available (10), and refrigeration is effective in checking the spread of decay (4,10). Some common bean cultivars are highly resistant to the disease.

1 ADEGBOLA M.O.K. & HAGEDORN D.J. (1969) Symptomatology and epidemiology of *Pythium* bean blight. *Phytopathology* **59**, 1113–1118 illus.
2 BHARGAVA K.S. & GUPTA S.C. (1957) Market diseases of fruits and vegetables in Kumaon. 1. Cottony leak of beans. *Indian Phytopathology* **10**, 48–49.
3 HARE W.W. (1949) Tip blight of garden pea. *Journal of Agricultural Research* **78**, 311–324.
4 HARTER L.L. & WHITNEY W.A. (1927) A transit disease of snap beans caused by *Pythium aphanidermatum*. *Journal of Agricultural Research* **34**, 443–447 illus.
5 KAGEYAMA K. & UI T. (1982) [Survival structure of *Pythium* spp. in the soils of bean fields.] *Annals of the Phytopathological Society of Japan* **48**, 308–313.
6 KIM S.H., KANTZES J.G. & WEAVER L.O. (1974) Infection of aboveground parts of bean by *Pythium aphanidermatum*. *Phytopathology* **64**, 373–380 illus.
7 LOCKE J.C., PAPAVIZAS G.C. & LEWIS J.A. (1979) Seed treatment with fungicides for the control of *Pythium* seed rot of peas. *Plant Disease Reporter* **63**, 725–728.
8 MAITI S. & SAHAMBI H.S. (1979) Aerial infection of *Pythium aphanidermatum* and *Corticium sasakii* on French bean in Meghalaya. *Indian Journal of Mycology and Plant Pathology* **9**, 99–100.
9 SEN B. & LAL S.P. (1969) Blight of broad bean – a new *Pythium* disease. *Plant Disease Reporter* **53**, 470–472.
10 WELLS J.M. & COOLEY T.N. (1973) Control of *Pythium* and *Sclerotinia* rots of snap beans with postharvest hot water and chemical dips. *Plant Disease Reporter* **57**, 234–236.

DOWNY MILDEW of peas and faba beans caused by

Peronospora viciae **(Berk.) Casp.**
(syn. *Peronospora pisi* Sydow)

143



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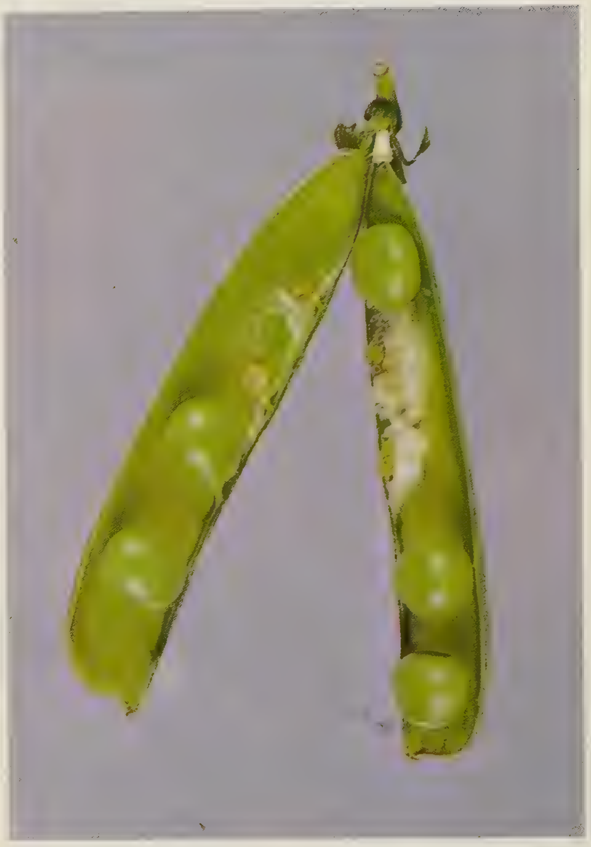
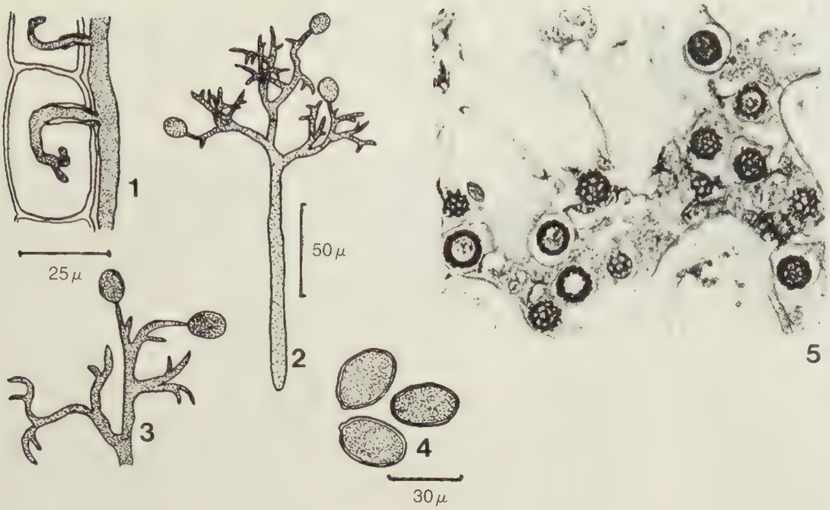


OCCURRENCE Downy mildew of peas is widespread, occurring in most countries in which the crop is grown (2). It is particularly common in areas possessing a cool maritime climate (1,2). Records include the UK (3,8), France (1), Belgium (10), Germany (4), the Netherlands, Denmark, Sweden (7), the USSR (6), northern India, the western USA (2,9) and Australia. Downy mildew of faba beans has been recorded in Tunisia (5) and is occasionally seen in the UK. The so-called 'downy mildew of lima bean' is not a downy mildew in the strict sense, and is considered under phytophthora rot (q.v.).

SYMPTOMS On faba bean pods small yellow spots appear (5). On pea pods yellowish blotches develop, gradually changing to a bright brown colour mottled with greenish-yellow islands (2). Inside the pod a white felt-like outgrowth of abnormal plant tissue may be seen, beneath which is an abundance of thick-walled spores (oospores) (1,2,9). Alternatively, or in addition, mould strands may spread over the inner surface of the pod and give rise to oospores, the mould appearing to the naked eye like a mealy layer, at first white and later brown (9). Seeds may be infected, and show small reddish marks (1) or brown sunken spots within brownish-grey discoloured areas (2). Seed-coat splitting may occur, resembling that commonly associated with pea seedborne mosaic virus (q.v.)

BIOLOGY The fungus survives in the soil in the sexual state (oospores) (1,7). In cool wet conditions these spores germinate and can cause infection of the stem tissue of pea seedlings (8). Subsequently the fungus invades all parts of the young plant (systemic infection) and, if the air remains cool and humid, there is copious production of asexual spores (conidia) on the lower surface of leaves (1,2). Such plants usually die. At moderate temperatures (below 20°C) and in the presence of moisture (rain or dew) the air-borne spores are capable of causing secondary infection (local or systemic) of older plants (7). Systemic infection via growing points may result in delayed pod development (8), while local infection of pods causes disfigurement (2). Pods intended for the fresh market may thus be reduced in value (2), while crops grown under contract for processing may fail to meet specifications relating to uniformity or date of harvest (8).



145 *Peronospora viciae* (from CMI Descr. No. 455, Mukerji 1975)
1 intercellular mycelium with haustoria 2 sporangiophore
3 branch of sporangiophore 4 sporangia 5 mycelium, haustoria, oogonia, antheridia and oospores × 100

CONTROL Crop rotation (5) may not be effective, since the fungus can survive for many years in the soil (7). It is therefore important to minimise soil infestation by prompt removal and destruction of infested crop debris. If a field is known to be contaminated, pea seed may be treated with a systemic fungicide, which continues to protect the seedlings as they develop (10). It may also be beneficial to spray the crop with a fungicide, especially at the onset of flowering (7); however, in the absence of effective disease forecasting systems (7), this does not always provide satisfactory control (1). Reduction of disease has been obtained by the use of a herbicide which, in addition to killing weeds, also kills systemically infected plants and the spores produced on them (7). Good drainage is important (5). Some pea cultivars are partially resistant to the disease (1,7), but breeding techniques have to take account of the fact that several races of this fungus exist (3,4). Although seed transmission does not appear to be of major importance (8), it has been demonstrated that the fungus may be carried to new areas in seed (1).

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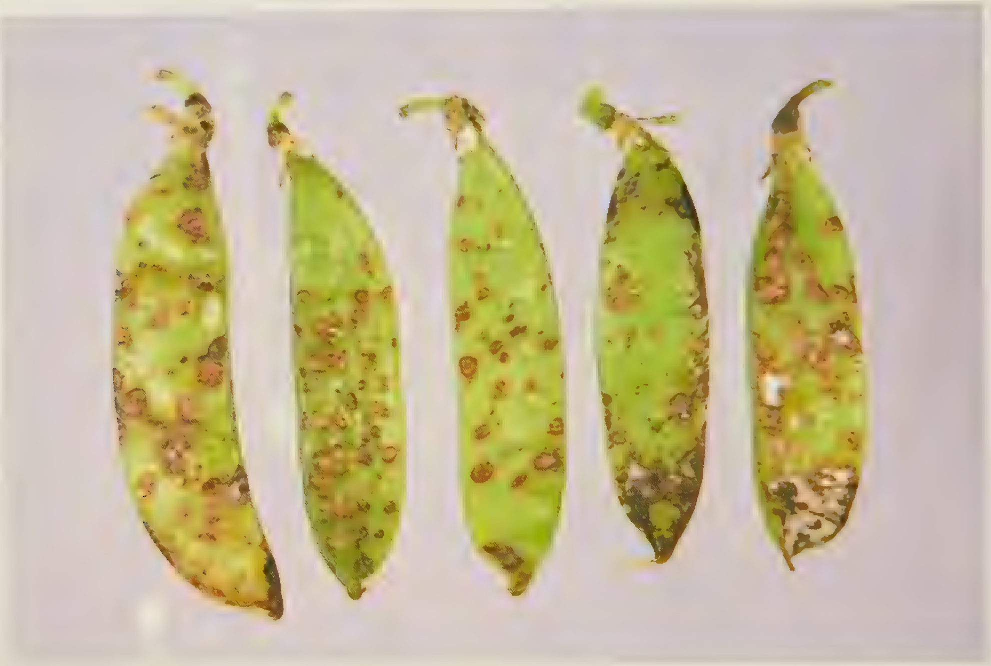
1 ALLARD C. (1970) Recherches sur la biologie du mildiou du pois. *Annales Phytopathologiques* **2**, 87–115 illus.
2 CAMPBELL L. (1935) Downy mildew of peas caused by *Peronospora pisi* (De B.) Syd. *Bulletin of Washington Agricultural Experiment Station* No. 318, 42 pp illus.
3 DICKINSON C.H. & SINGH H. (1982) Colonization and sporulation of *Peronospora viciae* on cultivars of *Pisum sativum. Plant Pathology* **31**, 333–341.
4 HEYENDORFF R.C. von & HOFFMANN G.M. (1978) Zur physiologischen Spezialisierung von *Peronospora pisi* Syd. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **85**, 561–569.
5 JAMOUSSI B. (1968) Attaque de mildiou sur fève. *Archives de l'Institut Pasteur, Tunis* **45**, 117–127 illus.
6 KIRIK N.N. & KOSHEVSKII I.I. (1977) [Features of the pathogenesis of downy mildew of pea.] *Mikologiya i Fitopatologiya* **11**, 332–335.
7 OLOFSSON J. (1966) Downy mildew of peas in western Europe. *Plant Disease Reporter* **50**, 257–261 illus.
8 PEGG G.F. & MENCE M.J. (1972) The biology of *Peronospora viciae* on pea: the development of local and systemic infections and their effect on vining yield. *Annals of Applied Biology* **71**, 19–31.
9 SNYDER W.C. (1934) *Peronospora viciae* and internal proliferation in pea pods. *Phytopathology* **24**, 1358–1365 illus.
10 VULSTEKE G. & MEEUS P. (1985) Control of *Peronospora viciae* (Berk.) de Bary f. sp. *pisi* in peas. *Mededelingen van de Faculteit Landbouwwetenschappen Rijksuniversiteit Gent* **50**(36), 1205–1215.

MYCOSPHAERELLA BLIGHT of peas and beans caused by

Mycosphaerella pinodes **(Berk. & Blox.) Vestergr.**

Conidial state: *Ascochyta pinodes* Jones

146

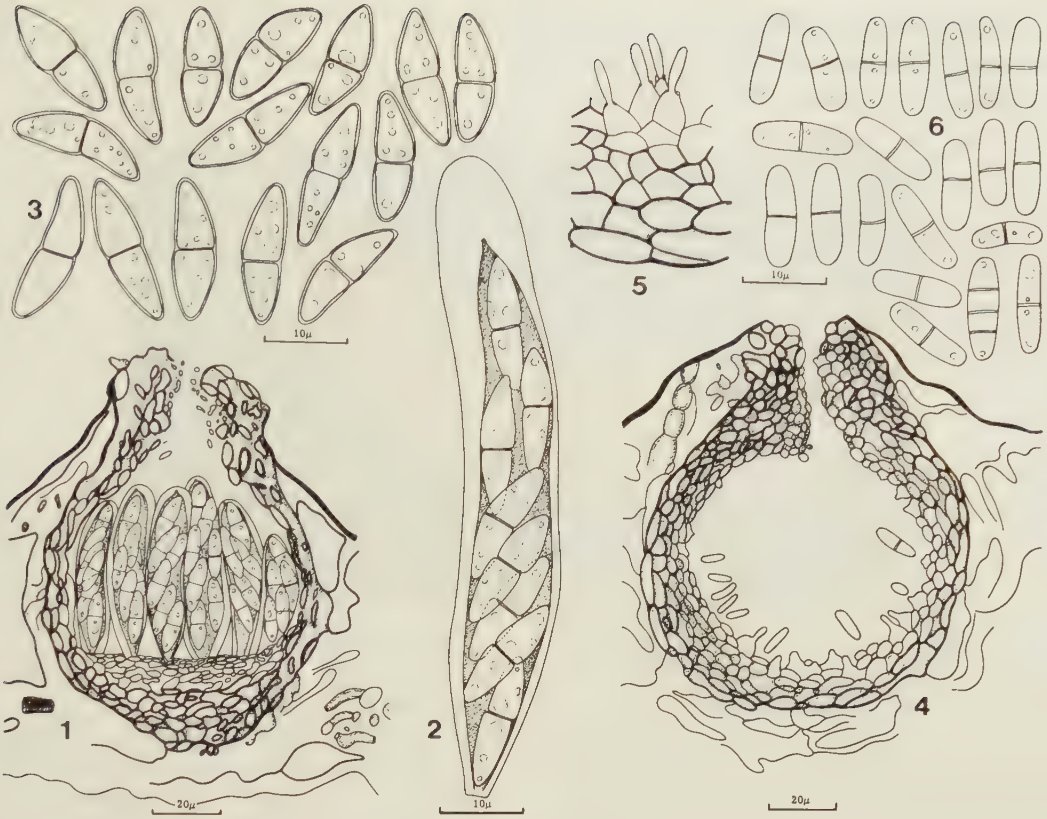


OCCURRENCE The fungus is widespread, especially in temperate and subtropical areas. Records include Canada (9), the USA (5), Chile, South Africa (10), Australia (3,4), Eire (8), the Netherlands (6), Belgium (2) and Germany (1). Mycosphaerella blight tends to be the most serious of three related pea diseases (5,9) which often occur together, the other two being ascochyta blight and pod spot (q.v.) caused by *Ascochyta pisi*, and foot rot (and occasional pod spot) caused by *Phoma medicaginis* var. *pinodella* (syn. *Ascochyta pinodella*). Although peas are the main host, faba beans and common beans are occasionally subject to mycosphaerella blight (1,7).

SYMPTOMS Pods become flecked with numerous small dark streaks and spots which may be purplish-black at first but which later turn reddish-brown (5). These lesions enlarge rapidly, and eventually minute spherical bodies (pycnidia) are produced in a circle or scattered within each lesion (10).

BIOLOGY In contrast to the related species mentioned above, *Mycosphaerella pinodes* possesses a sexual stage (pseudothecia giving rise to ascospores), the importance of which appears to vary in different localities (4,8). In this form, and also as resting structures (chlamydospores and sclerotia), the fungus is capable of surviving adverse conditions (8). Thus, in addition to being carried in seed, it remains viable in crop debris through the winter in Europe, for example (6), and through the dry summer in South Australia (4). With the onset of rain or irrigation, coupled with moderate temperatures, ascospores are released into the air and are capable of direct infection of moist leaves, stems and pods (1,4,6,7). The asexual stage (pycnidia giving rise to conidia) is produced in the lesions within a few days, and new infections are caused by waterborne spores (5). As the lesions mature and the infected organ becomes moribund, the sexual stage usually develops, so completing the cycle (1,10). Cold or dry conditions ensure that ascospore development is delayed until the next growing season, when the new crop is liable to be infected (6). Even if conditions permit early ascospore release, the disease may be perpetuated by infection of unharvested 'volunteer plants' (6).

CONTROL Pea plants should be burned or else ploughed in deeply as soon as the crop has been harvested. Since in some soils the fungus may nevertheless persist for several years (8), a long rotation with other crops has also been recommended. Because ascospores can be blown considerable distances (4), fields selected for a new crop should not be adjacent to land which has recently been planted with peas. Clean seed may be obtained from dry growing areas where the disease



147 *Mycosphaerella pinodes* (from CMI Descr. No. 340, Punithalingam & Holliday 1972) 1 pseudothecium 2 ascus 3 ascospores 4 pycnidium 5 part of pycnidial wall and conidiophores 6 conidia

is rare; otherwise seed should be treated with a fungicide (3,6,9). It has been demonstrated that plants may be protected from mycosphaerella blight by pre-inoculation of the seedlings with the related fungus *A. fabae* (2). More usually, however, the growing crop is sprayed with an appropriate fungicide (3).

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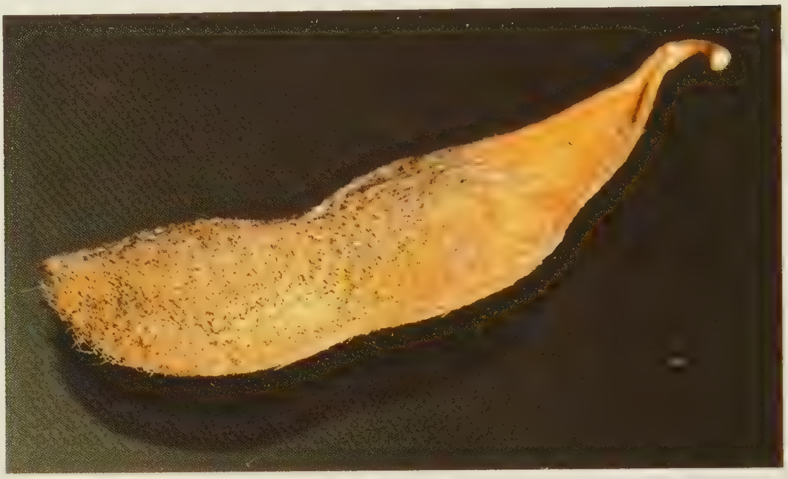
1 BAUMANN G. (1953) Untersuchungen zur Biologie von *Mycosphaerella pinodes* (Berk. et Blox.) Stone. *Kühn-Archiv* **67**, 305–383 illus.
2 BOY N. (1981) Protection locale du pois contre *Mycosphaerella pinodes* (Berk. et Blox.) par préinoculation avec *Ascochyta fabae* (Speg.). *Parasitica* **37**, 109–118.
3 BRETAG T.W. (1985) Chemical control of *Ascochyta* blight of field peas. *Australasian Plant Pathology* **14**(2), 42–43.
4 CARTER M.V. (1963) *Mycosphaerella pinodes*. 2. The phenology of ascospore release. *Australian Journal of Biological Sciences* **16**, 800–817.
5 HARE W.W. & WALKER J.C. (1944) Ascochyta diseases of canning pea. *Research Bulletin of Wisconsin Agricultural Experiment Station* No. 150, 31 pp illus.
6 KERLING L.C.P. (1949) Aantasting van erwten door *Mycosphaerella pinodes* (Berk. et Blox.) Stone. *Tijdschrift over Plantenziekten* **55**, 41–68 illus.
7 SATTAR A. (1933) A comparative study of the fungi associated with blight diseases of certain cultivated leguminous plants. *Transactions of the British Mycological Society* **18**, 276–301 illus.
8 SHERIDAN J.J. (1973) The survival of *Mycosphaerella pinodes* on pea haulm buried in soil. *Annals of Applied Biology* **75**, 195–203.
9 WALLEN V.R., CUDDY T.F. & GRAINGER P.N. (1967) Epidemiology and control of *Ascochyta pinodes* on field peas in Canada. *Canadian Journal of Plant Science* **47**, 395–403 illus.
10 WARMELO K.T. van (1966) The identity of a pea blight fungus in South Africa. The cytology of *Mycosphaerella pinodes*. *Bothalia* **9**, 183–194 illus.

PHOMOPSIS POD BLIGHT of beans caused by

***Diaporthe phaseolorum* (Cooke & Ellis) Sacc.**

Conidial state: *Phomopsis phaseoli* (Desm.) Grove

148



OCCURRENCE An unspecified *Diaporthe* has been recorded on seed of common beans in Brazil (3). *D. phaseolorum* var. *phaseolorum* occurs on lima bean, and has caused losses in the southern and eastern USA (4,7), Cuba, Bermuda, South Africa and Tanzania. *D. phaseolorum* var. *sojae* has a wider host range including common bean, lima bean and soybean. Most of the published work is on soybean (1,2,5,6, 7,9,10), and the fungus causing pod and stem blight of this host is often referred to as *Phomopsis sojae* Lehman. A detailed study concluded that all of these are synonymous (6).

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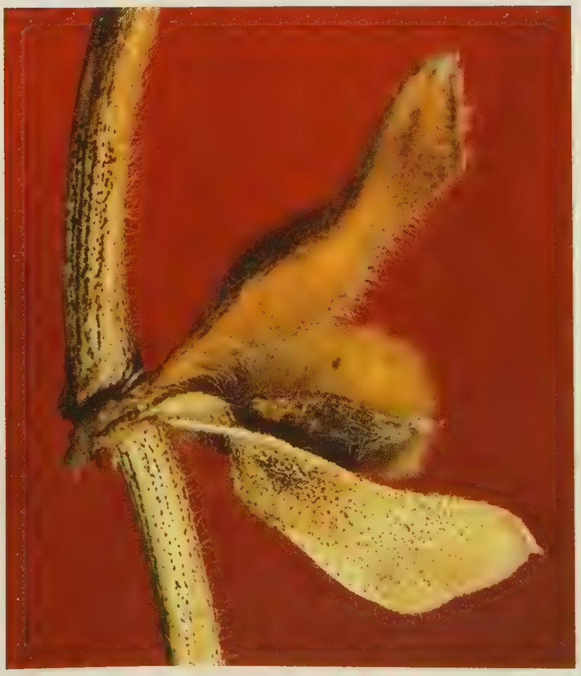
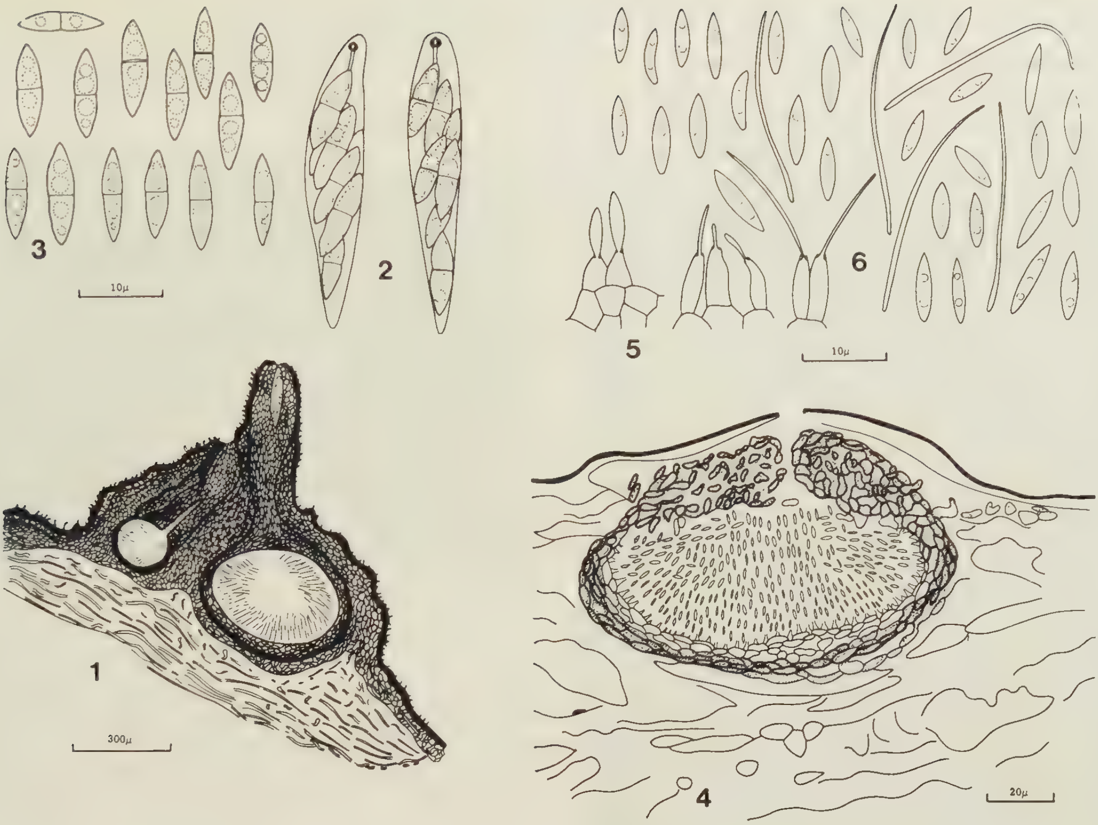


SYMPTOMS Reddish-brown lesions may form on any part of the pod but most commonly occur along the sutures (4). Minute black bodies (pycnidia) develop, often in concentric circles, within the lesions. Subsequently, as the pod becomes generally moribund, these structures are more likely to be produced in a random manner throughout the infected tissue (4); such a symptom may also occur on kidney beans (7). The seed in dried pods may be infected and shrivelled even when there is no evidence of the fungus on the exterior of the pods (7).

BIOLOGY The fungus survives in crop debris (4) and can persist in soil. It may produce the sexual stage (perithecia giving rise to ascospores) as well as the asexual stage (pycnidia giving rise to conidia). Warm wet conditions favour spore dispersal and permit direct infection of leaves, stems and, later, pods, whether immature (7) or mature (4). The likelihood of infection is further increased if the plants have been injured, for example by high winds or by workers moving through the crop. Blemished pods are reduced in value; furthermore, decay can continue after harvest, the optimal temperature for fungal growth being between 25° and 30°C (4). Seed may become infected (3,4,7).

CONTROL Pod blight of lima beans has declined in importance now that seed crops are mostly grown in dry areas. If clean seed is not available, a fungicide seed treatment should be used (4). Spraying the crop can also be effective if started early enough (4). If pods are found to be infected they should be cooled immediately after harvest and marketed without delay.



150 *Diaporthe phaseolorum* (from CMI Descr. No. 336, Punithalingam & Holliday 1972) 1 perithecia 2 asci 3 ascospores 4 pycnidium 5 conidiophores 6 conidia

1 ATHOW K.L. & CALDWELL R.M. (1954) A comparative study of *Diaporthe* stem canker and pod and stem blight of soybean. *Phytopathology* **44**, 319–325 illus.

2 BALDUCCHI A.J. & McGEE D.C. (1987) Environmental factors influencing infection of soybean seeds by *Phomopsis* and *Diaporthe* species during seed maturation. *Plant Disease* **71**, 209–212.

3 FULCO W. da S., CAMARGO M.R. de O., ALTMAYER M.B. & BRUSAMOLIN E.P. (1977) Análise sanitária em sementes de três cultivares de feijão (*Phaseolus vulgaris* L.) procedentes de dois municípios do Rio Grande do Sul. *Agronomia Sulriograndense* **13**, 361–367.

4 HARTER L.L. (1917) Pod blight of the lima bean caused by *Diaporthe phaseolorum*. *Journal of Agricultural Research* **11**, 473–504 illus.

5 HILDEBRAND A.A. (1956) Observations on stem canker and pod and stem blight of soybeans in Ontario. *Canadian Journal of Botany* **34**, 577–599 illus.

6 KULIK M.M. (1984) Symptomless infection, persistence, and production of pycnidia in host and non-host plants by *Phomopsis batatae, Phomopsis phaseoli*, and *Phomopsis sojae*, and the taxonomic implications. *Mycologia* **76**, 274-291.

7 LUTTRELL E.S. (1947) *Diaporthe phaseolorum* var. *sojae* on crop plants. *Phytopathology* **37**, 445–465 illus.

8 MOORE W.D. (1931) Pod blight of lima beans in North Carolina and Virginia. *Plant Disease Reporter* **15**, 113–114.

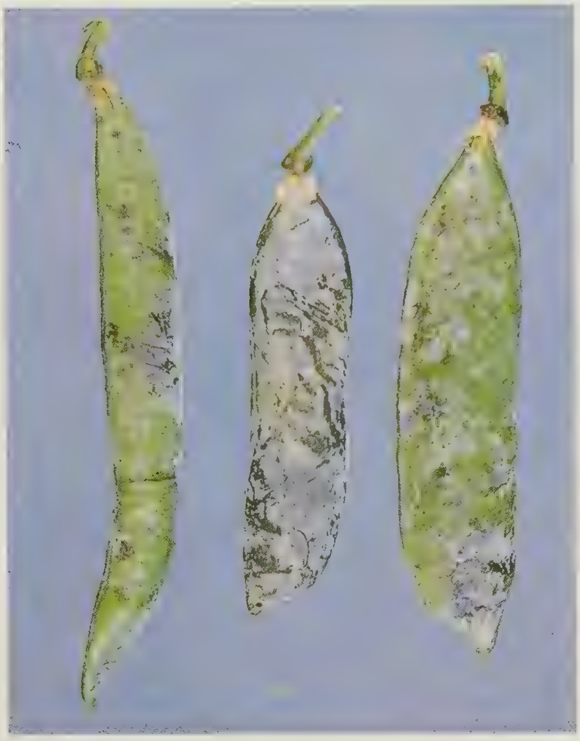
9 RUPE J.C. & FERRISS R.S. (1986) Effects of pod moisture on soybean seed infection by *Phomopsis* sp. *Phytopathology* **76**, 273–277.

10 SINCLAIR J.B. (Ed.) (1989) *Compendium of soybean diseases*. 3rd ed. Saint Paul, Minnesota: American Phytopathological Society, 106 pp illus.

POWDERY MILDEW of peas and beans caused by

***Erysiphe* spp.**
Conidial state: *Oidium* spp.
***Microsphaera penicillata* (Wall. ex Fr.) Lev.**

151



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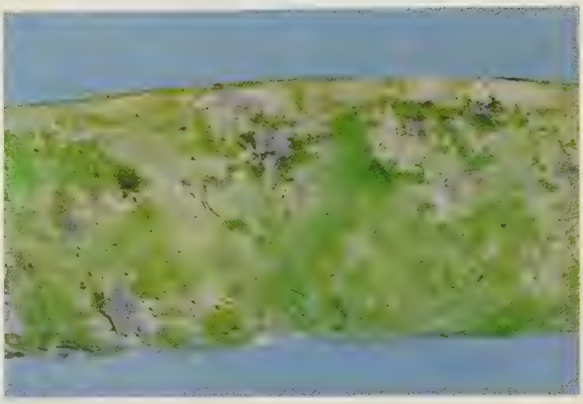
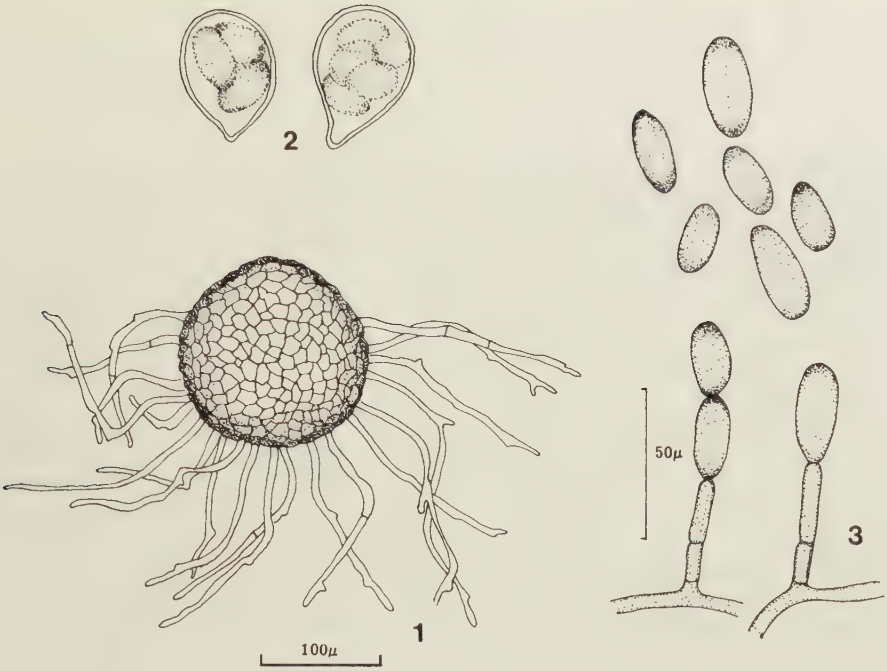


OCCURRENCE Powdery mildew of peas, caused by *Erysiphe pisi* DC. (syn. *Erysiphe polygoni* DC.) occurs wherever the crop is grown, for example the UK (2), the USA (8), China (10) and Argentina (6). It is of particular importance in parts of India (3,7,9). The disease is occasionally recorded on faba beans, the causal organism being *E. pisi* in Argentina (6), India (9) and China (10), and *Microsphaera penicillata* in parts of Canada (4). Powdery mildew of common beans and lima beans is usually caused by *E. pisi* (5,8); the fungus is especially virulent in the Caribbean, where it attacks common bean cultivars considered resistant in the USA. *E. cichoracearum* (page 205) has been recorded on common beans in Germany (1).

SYMPTOMS On mature pods the first symptoms are small circular spots, slightly discoloured in peas (6), reddish in faba beans (10), and 'moist-looking' in common beans (5). These enlarge into extensive irregular reddish-brown lesions which at some stage may be covered with a whitish powdery mould bearing abundant spores (conidia) (5). Infected seeds show a greyish-brown discoloration.

BIOLOGY These fungi may produce the sexual state (cleistothecia giving rise to ascospores) (4) as a means of surviving adverse conditions. More commonly, however, they exist in the asexual state (conidia) on fresh infected tissue of peas, beans and certain weeds (4,5,10), though they do not form on non-living debris. These spores can be blown great distances in the atmosphere and remain viable. In contrast to the spores of most other fungi, powdery mildew conidia are able to germinate without any external supply of moisture (2). Direct penetration occurs, and infection is favoured by warm dry weather with occasional showers or dew formation; disease development is inhibited by heavy or prolonged rainfall (5,6,9). Lesions first appear on the upper surface of leaves, but may later be present on both surfaces, as patches of white mould; minute black bodies (cleistothecia) may eventually be produced (4). Infection of stems and pods also occurs, and in some regions the disease often reaches epidemic proportions (5,9). Although essentially a field disease causing a reduction in yield, powdery mildew may be responsible for post-harvest losses as a result of pod blemishes and poor quality seed.



153 *Erysiphe pisi* (from CMI Descr. No. 155, Kapoor 1967) 1 cleistothecium 2 asci and ascospores 3 conidia and conidiophores

CONTROL Cultivars differ in their resistance to powdery mildew (3,5,6), and breeding work continues, complicated by the existence of numerous races of the fungi (8,10). Most of these races are host-specific, but there are reports of *E. pisi* from pea infecting faba bean (and *vice versa*) (10) and *E. cichoracearum* from bean infecting cucumber (1). Since seed-transmission has been demonstrated, at least in peas (9), it is desirable to sow healthy or treated seed. However, since spores may nevertheless be present in the atmosphere, clearly it could be of equal or greater importance to apply fungicide to the growing crop (5,6,9). In some localities effective control is achieved by the judicious use of overhead sprinkler irrigation which inhibits powdery mildew development.

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1 BEICHT W. & WESSEL H. (1983) Beobachtungen zum Auftreten eines Echten mehltaupilzes an Bohnen. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **90**, 291–294 illus.

2 GAY J.L., MARTIN M. & BALL E. (1985) The impermeability of powdery mildew conidia and their germination in arid environments. *Plant Pathology* **34**, 353–362 illus.

3 KALIA P. (1985) Pea varieties resistant to powdery-mildew. *Indian Journal of Agricultural Sciences* **55**, 206–208.

4 KHARBANDA P.D. & BERNIER C.C. (1977) Powdery mildew of *Vicia faba* in Manitoba. *Canadian Journal of Plant Science* **57**, 745–749 illus.

5 MOORE W.D. (1936) Powdery mildew (*Erysiphe polygoni*) on garden and snap beans. *Phytopathology* **26**, 1135–1144 illus.

6 PLOPER L.D. (1981) El oidio de la arveja (*Pisum sativum* L. var. *arvense* (L.) Poir) en Tucumán. *Revista Industrial y Agrícola de Tucumán* **58**(1), 69–79 illus.

7 SHUKLA P. & LAL S.S. (1988) Effect of combined application of fungicides and insecticides on the powdery mildew and pod borer of field pea. *Pesticides* **22**(3), 5–7.

8 STAVELY J.R. & HANSON E.W. (1966) Pathogenicity and morphology of isolates of *Erysiphe polygoni*. *Phytopathology* **56**, 309–318 illus.

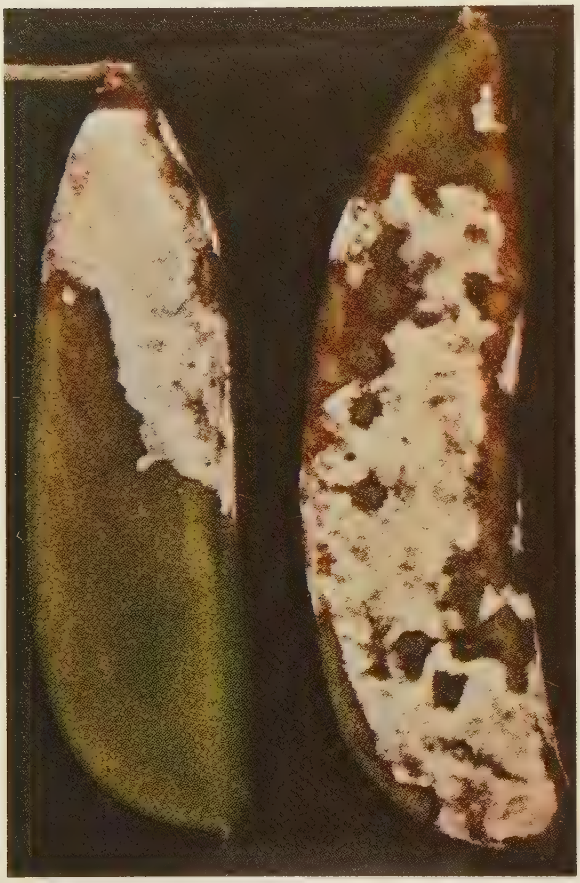
9 UPPAL B.N., PATEL M.K. & KAMAT M.N. (1935) Pea powdery mildew in Bombay. *Bulletin of the Department of Agriculture, Bombay* No. 177, 12 pp illus.

10 YU T.F. (1946) Powdery mildew of broad bean caused by *Erysiphe polygoni* DC. in Yunnan, China. *Phytopathology* **36**, 370–378.

PHYTOPHTHORA ROT of beans caused by

Phytophthora nicotianae **van Breda de Haan var.** ***parasitica*** **(Dastur) Waterh.**
Phytophthora phaseoli **Thaxter**

154



OCCURRENCE *Phytophthora nicotianae* var. *parasitica* has been recorded on common beans in Puerto Rico and in India (4). *P. phaseoli* may infect common bean (1) but occurs chiefly on lima beans, causing an important disease which is usually referred to as 'downy mildew' (q.v.). Most reports are from the eastern and central USA (2,3,5), but the disease also occurs in Canada, Mexico (1), the Philippines, Sri Lanka, Zaire, the USSR and Italy (CMI Map 201).

SYMPTOMS Pods initially show watersoaked patches, which soon support the growth of a pure white mould (4,5); this may be either loose and cottony or dense and felt-like, according to the environment in which it developed. Eventually the decaying pods shrivel; seeds may also be infected. Both fungi produce abundant thick-walled spores (oospores) in the infected tissue of pods and seeds (4,5).

BIOLOGY Both fungi can survive adverse conditions in the sexual state (oospores) (1,2,5). They can also persist as vegetative resting spores (chlamydospores) (1,4). Wet weather favours development of the asexual state (sporangia) (1). Sporangia of both fungi can give rise to zoospores, though sometimes sporangia of *P. phaseoli* germinate directly (2). Seedlings are particularly susceptible to infection, but mature plants may also be attacked if wet conditions prevail (3). Splash dispersal from infested soil or debris means that pods hanging near the ground are especially vulnerable (4). Infection can occur by direct penetration of sound tissue, though the presence of injuries is a predisposing factor (4). *P. nicotianae* var. *parasitica* can spread into neighbouring sound beans in store (4). In contrast, it is reported that *P. phaseoli* does not appear to develop further after harvest.

CONTROL Prompt removal and destruction of crop debris serves to minimise the level of infestation in the field. Phytophthora rot of common beans could probably be held in check by fungicide sprays (4) or, in small plots, by a mulch to prevent soil splash. 'Downy mildew' of lima bean can be controlled by timely fungicide sprays, and sophisticated forecasting systems have been devised, based on meteorological data for particular localities (2,3). Hot dry weather inhibits disease development and precludes the necessity for protective sprays (2,3). Breeding for resistance has been successful despite the occasional emergence of new races of *P. phaseoli*.

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1 BUENROSTRO O. & SORIANO E. (1987) Identificación de *Phytophthora phaseoli* a partir de lesiones en *Phaseolus vulgaris*. *Revista Mexicana de Fitopatología* **5**, 98–100 illus.

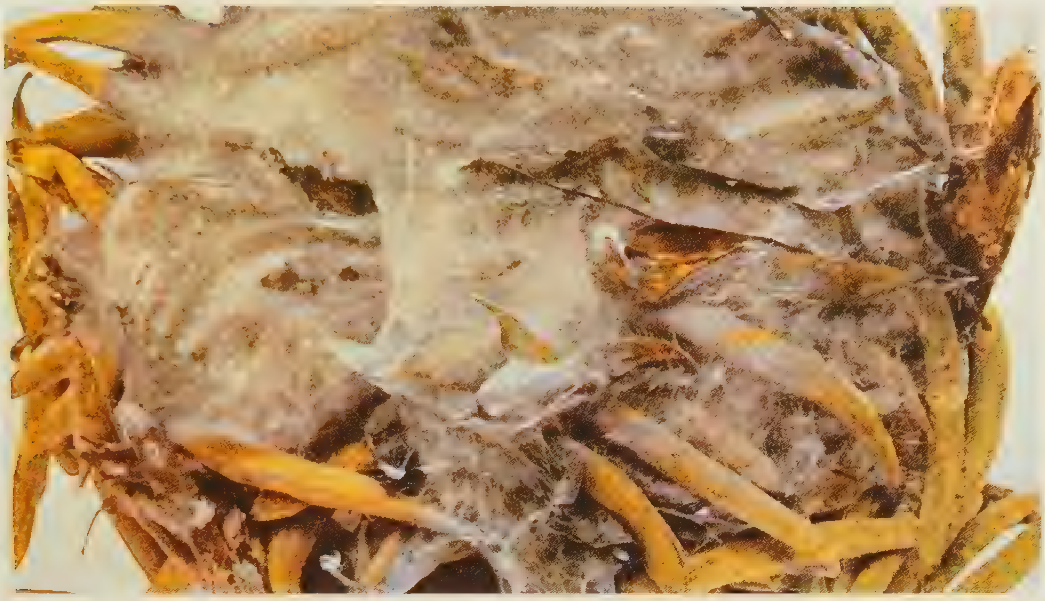
2 HYRE R.A. & COX R.S. (1953) Factors affecting viability and growth of *Phytophthora phaseoli*. *Phytopathology* **43**, 419–425 illus.

3 SCARPA M.J. & RANIERE L.C. (1964) The use of consecutive hourly dewpoints in forecasting downy mildew of lima bean. *Plant Disease Reporter* **48**, 77–81.

4 VENKATAKRISHNAIYA N.S. (1950) *Phytophthora parasitica* on French bean, *Phaseolus vulgaris* Linn. *Indian Journal of Agricultural Science* **20**, 391–394 illus.

5 WESTER R.E., GOTH R.W. & DRECHSLER C. (1966) Overwintering of *Phytophthora phaseoli*. *Phytopathology* **56**, 95–97 illus.

RHIZOPUS ROT of peas and beans caused by
Rhizopus oryzae **Went & Prinsen Geerligs**
Rhizopus stolonifer **(Ehrenb. ex Fr.) Lind**



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OCCURRENCE Rhizopus rot may occur in peas and all types of green beans, if environmental conditions permit (3). The disease has been reported on faba bean pods in the field in Romania (4), on stored pea pods in India (2) and on snap bean pods in transit from the southern states of the USA (3,5). The two species often occur together (3). *R. stolonifer* has been found in pea and bean seeds in Saudi Arabia (1).

SYMPTOMS Decayed tissue is typically soft and watery, with considerable leakage of fluid (5). In contrast to cottony leak (q.v.) or watery soft rot (q.v.), rhizopus rot is characterised by the production of coarse white mould strands and globular black spore heads (sporangia) (5). There may also be a distinctive sour odour, at least in the early stages of decay.

BIOLOGY *R. oryzae* may persist as resting spores (chlamydospores) and *R. stolonifer* occasionally produces the sexual state (zygospores). However, both fungi more commonly exist in the asexual state (sporangia giving rise to sporangiospores). Both species are ubiquitous in air, soil and water (3,5) and may be present on materials used for packaging (2); thus the pods may become contaminated at any time. Injury predisposes pods to infection, which occurs in moist conditions (2,5). Temperature is the predominant factor, however, rhizopus rot occurring only under warm conditions (3). 'Nests' of decay develop as neighbouring healthy pods are invaded (5); seeds may also be infected (1).

CONTROL Careful handling is necessary to prevent injury to the pods and, if they are to be held at ambient temperature, moisture should be excluded from the packs (5). Good control can be achieved by rapid pre-cooling, followed by refrigerated storage (2) and transport (3), since temperatures of 8° to 10°C inhibit the development of rhizopus rot (3,5).

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1 ABDEL-HAFEZ S.I.I. (1984) Mycoflora of bean, broad bean, lentil, lupine and pea seeds in Saudi Arabia. *Mycopathologia* **88**, 45–49.

2 BHATNAGAR D.K., THAKRAL K.K., SINGH J.P. & KALLOO (1984) Storage studies in pea (*Pisum sativum* L.) as influenced by packing material. *Journal of Research of Haryana Agricultural University* No. 14, 318–323.

3 LAURITZEN J.I., HARTER L.L. & WHITNEY W.A. (1933) Environmental factors in relation to snap bean diseases occurring in shipment. *Phytopathology* **23**, 411–445.

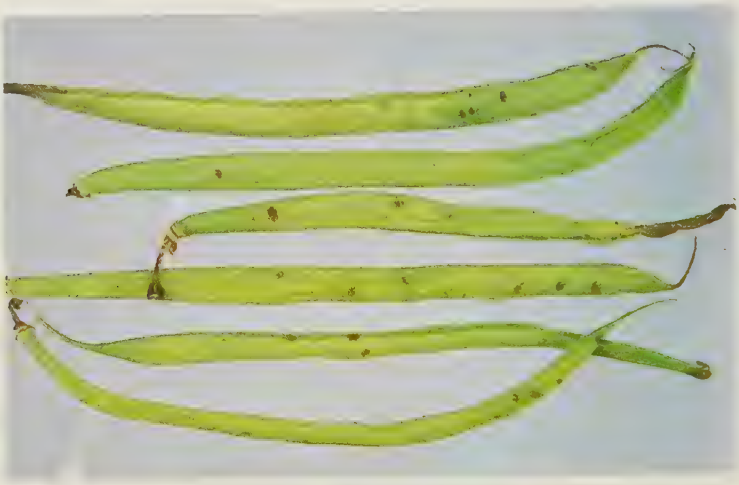
4 PUŞCAŞU A. (1986) [Pod rot – a new disease of broad beans recorded in Romania.] *Analele Institutului de Cercetări pentru Protecţia Plantelor* **19**, 81–84.

5 RAMSEY G.B. & WIANT J.S. (1941) Market diseases of fruits and vegetables: asparagus, onions, beans, peas, carrots, celery, and related vegetables. *Miscellaneous Publication of the United States Department of Agriculture* No. 440, 70 pp illus.

RUST of beans caused by

Uromyces appendiculatus **(Pers.) Unger**
(syn. *Uromyces phaseoli* (Pers.) Wint.)

156



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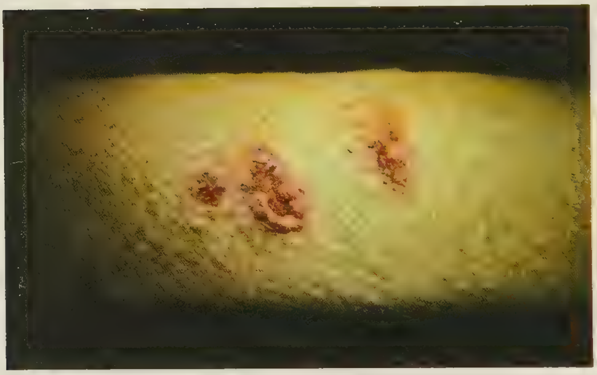
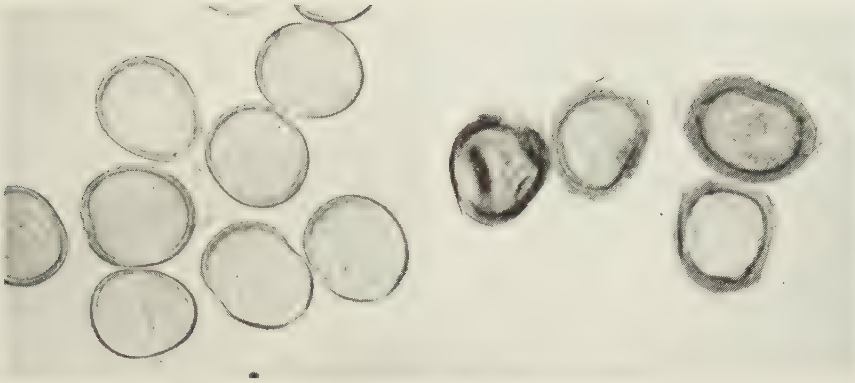


OCCURRENCE Bean rust has worldwide distribution (2) and occurs in most bean-growing areas except those with a dry climate (CMI Map 290). Records of rust on common beans include the USA (2,4,5,10), Costa Rica (6), Kenya (9), Germany (1), Australia (7) and New Zealand (3). The disease is of minor importance on lima beans.

SYMPTOMS Pod lesions begin as brown spots which later erupt into pustules (up to 2 mm across) containing dark brown to black spores (3).

BIOLOGY This fungus may produce several types of spore, all of which are associated with beans (5). Sometimes on the upper surface of leaves there may be numerous pale raised lesions (spermagonia giving rise to spermatia), whilst at corresponding locations on the underside there later develop clusters of white pustules (aecia giving rise to aeciospores). Subsequent infection of leaves by wind-blown aeciospores results in the formation of spots which develop into brown pustules (uredinia giving rise to urediniospores) (5). These 'summer spores' are also dispersed by wind and, under moist conditions, can cause infection of leaves and pods, resulting in more brown pustules (3). Cloudy humid weather favours disease development and, if moderate temperatures and high humidities prevail, the disease may become epidemic (1,3). The brown pustules subsequently turn into black pustules (telia giving rise to teliospores), and these 'winter spores' survive on bean debris and on poles used for staking the plants (3). After a rest period they are capable of germinating in the presence of moisture to produce basidiospores, which may cause infection of the next crop (5). All these stages have been observed in parts of the USA and in the Netherlands (Hubbeling, page 99). In warm regions of the world, however, the fungus may exist solely as summer spores, surviving from season to season. The first parts of the plant to be infected are usually the lower leaves (3), invasion taking place via the natural openings



158 *Uromyces appendiculatus* (from CMI Descr. No. 57, Laundon & Waterston 1965)
Urediniospores and teliospores × 600

(stomata) (1). Affected plants are predisposed to secondary infection by other disease organisms such as *Ascochyta* spp. (q.v.). Severe rust attacks can result in defoliation, stunted growth and consequent reduced yield (9). Pods infected shortly before harvest may be rejected in the market because of the development of disfiguring lesions (7).

CONTROL Crop rotation is desirable, and it may be beneficial to grow beans in association with another crop such as maize (6). Although the fungus is not seed-borne, seed-treatment with a systemic fungicide has been suggested as a means of protecting developing seedlings (7). Wide spacing of plants assists in aeration and drying off, thereby reducing the risk of rust infection (7). The developing crop should be sprayed regularly (7,9,10). As an alternative to fungicides, biological control using an antagonistic bacterium is partially successful (2). After harvest, bean debris should be burned or ploughed in deeply (3,7), and poles disinfected either by flaming or by chemical treatment (3). Much effort has been put into the breeding of rust-resistant bean cultivars, and several are available (4,10). It is a continuing task, however, because of the existence of numerous races of the fungus which are constantly evolving (2,7,9).

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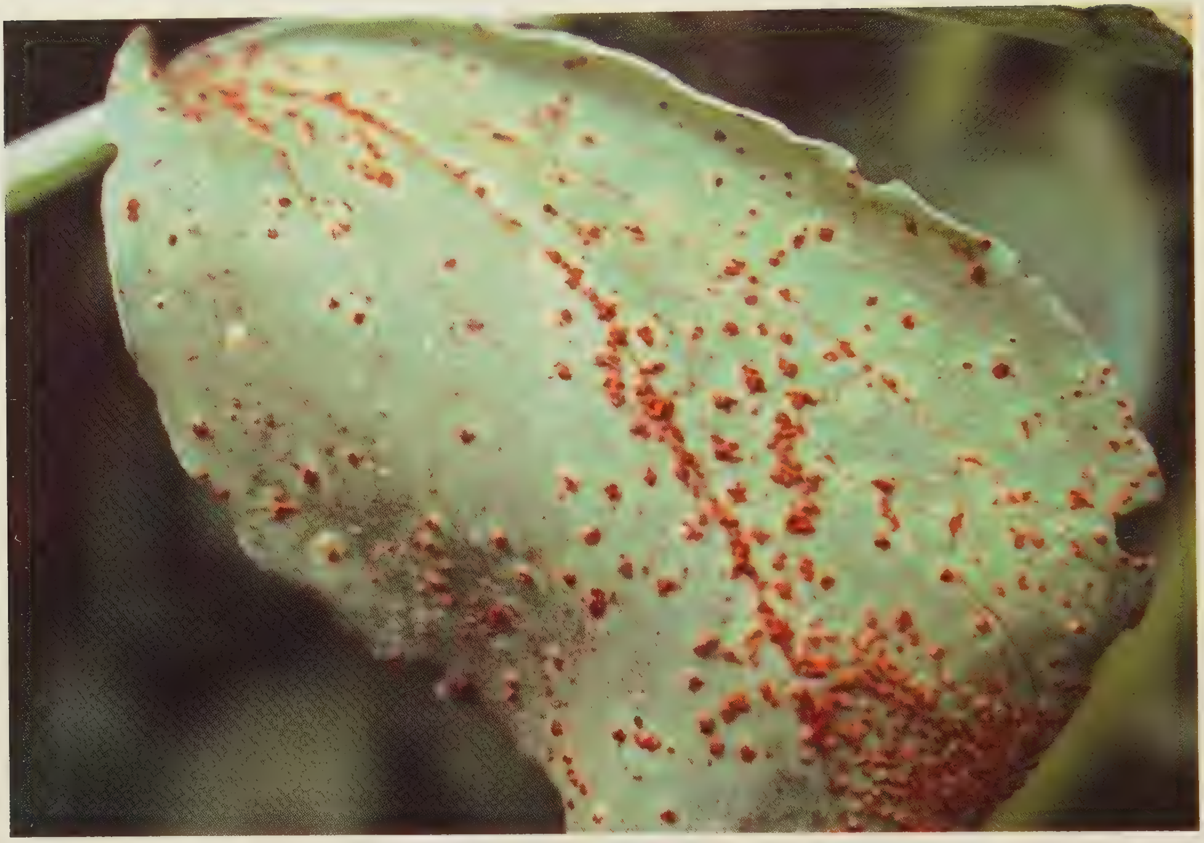
See also:

STAVELY J.R., STEADMAN J.R. & McMILLAN R.T. (1989) New pathogenic variability in *Uromyces appendiculatus* in North America. *Plant Disease* **73**, 428–432.

RUST of peas and faba beans caused by

Uromyces pisi-sativi (Pers.) Liro
Uromyces viciae-fabae (Pers.) Schröter

159

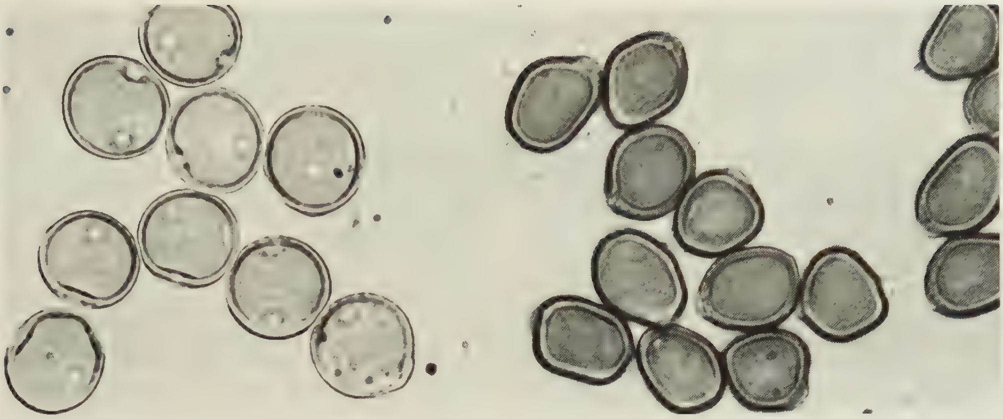


OCCURRENCE Several races of these fungi exist, some of which are able to infect both peas and faba beans (1,3,4). *Uromyces pisi-sativi* has been recorded in many European countries, for example Norway (4), and also occurs in North Africa, Asia and South America (CMI Map 404). *Uromyces viciae-fabae* is also widely distributed (CMI Map 200), for example it has been reported on peas in Canada (1), Norway (4) and India (8), and on faba beans in Canada (7), France, Belgium (2), Germany (3,5), the UK (10) and Egypt (6).

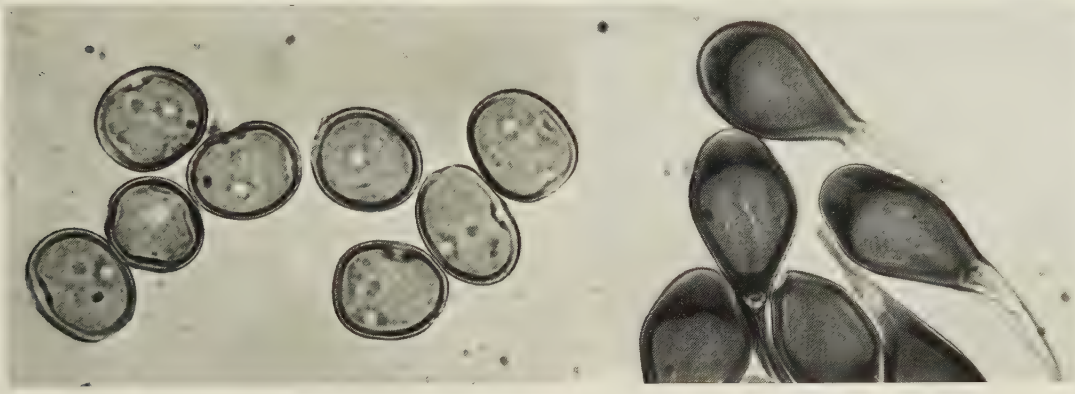
SYMPTOMS Brown lesions on the pods develop into raised pustules (up to 1 mm across) which split open to reveal quantities of brown spores (urediniospores).

BIOLOGY Each of the fungi produces several different types of spore. Both species produce 'summer spores' (urediniospores in uredinia) followed by 'winter spores' (teliospores in telia) on leaves, stems and pods of peas and faba beans. *U. pisi-sativi* then infects species of spurge (e.g. *Euphorbia cyparissias* L.), its alternate host, on which it produces spermagonia and aecia (3,4), while *U. viciae-fabae* completes its entire life cycle on the pea or bean (9). In warm climates aeciospores probably remain viable for some time (5) and serve to initiate infection in pea and bean crops. On the other hand, to survive winter in Norway or summer in India, these fungi probably persist as mould strands or teliospores (which subsequently germinate to give infective basidiospores, so completing the life-cycle). Infection is favoured by warm moist conditions (6), and severe attacks, besides reducing yields (10), may also result in the appearance of blemished pods in the market.

CONTROL It has been suggested that eradication of spurge plants would serve to control *U. pisi-sativi*. Since *U. viciae-fabae* has been recorded on seed of faba bean, it may be advisable to treat seed with a fungicide. Spraying the crop has also been recommended (2,8,10). Work is in progress to breed more rust-resistant pea and bean cultivars (7).



160 *Uromyces pisi-sativi* (from CMI Descr. No. 58, Laundon & Waterston 1965) Urediniospores and teliospores × 600



161 *Uromyces viciae-fabae* (from CMI Descr. No. 60, Laundon & Waterston 1965) Urediniospores and teliospores × 600

1 CONNER R.L. & BERNIER C.C. (1982) Host range of *Uromyces viciae-fabae. Phytopathology* **72**, 687–689.

2 EVRARD P. & LEPOIVRE P. (1983) Effet du dichlorure de triphénylbismuth vis-à-vis de la rouille du haricot et de la rouille de la féverole. *Parasitica* **39**, 117–127.

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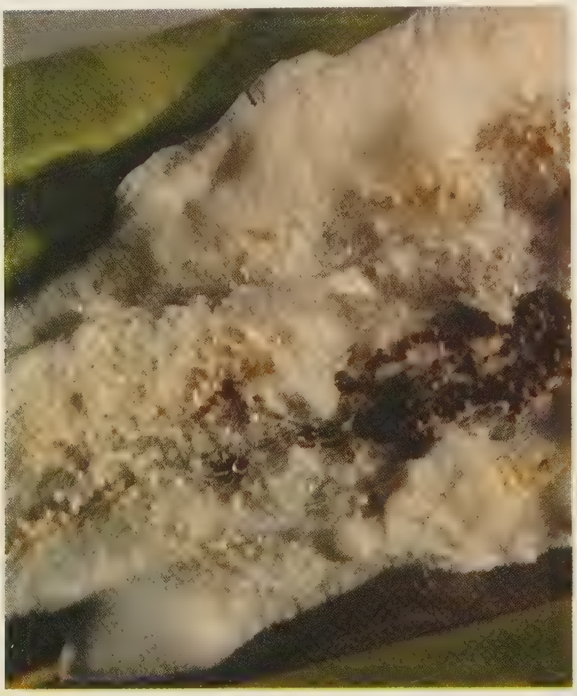
SCLEROTIUM ROT of peas and beans caused by

Corticium rolfsii **Curzi**

Sclerotial state: *Sclerotium rolfsii* Sacc.

162



OCCURRENCE In the field this disease is usually known as southern blight. It occurs in tropical and subtropical countries, and has been recorded on common beans in, for example, the southern USA (5), Mexico, Brazil, Venezuela, Cuba (1), Israel (4) and Australasia (6). Lima beans and faba beans are also susceptible (2). The disease does not usually affect peas (2), although there are occasional records from the southern USA.

SYMPTOMS Affected tissue is at first watersoaked but not discoloured. A white silky mould spreads over the pods in a characteristic fan-shaped pattern, and 'nests' of decay develop. Numerous small (1 to 2 mm) white resting bodies (sclerotia) are produced and turn brown within a few days (5).

BIOLOGY The fungus survives in the soil as sclerotia which, during warm wet weather, give rise to a mass of mould strands (5). These grow through the soil and, if moisture persists, are capable of attacking the base of the stem and also any plant parts in contact with the ground. Pods with incipient infection may be harvested in apparently good condition, only to decay during transit and storage if temperatures permit. The optimal growth temperature for the fungus is approximately 30°C and it can make substantial development at 20°C.

CONTROL Complete control is difficult, but there are several ways of reducing losses. The use of a mulch on the soil surface has the effect of delaying the onset of disease so that a reasonable yield of pods may be harvested (6). Diseased plants should be removed and destroyed. In countries with a hot dry season it is beneficial (between crops) to cover the field with polyethylene film, and make use of solar heating to raise the soil temperature (solarization) (3). Sclerotia are thereby killed or else weakened sufficiently to hasten natural decay before the planting of the new crop. Biological control entails the addition to the soil of preparations of certain fungi (1) or bacteria (4) which possess antagonistic or competitive properties, thus helping to reduce the incidence of disease. Control of post-harvest rotting is achieved by the use of refrigeration; at 8°C growth of the fungus is completely checked. Some bean cultivars (1) and most pea cultivars (2) are resistant to the disease.

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2 EPPS W.M., PATTERSON J.C. & FREEMAN I.E. (1951) Physiology and parasitism of *Sclerotium rolfsii. Phytopathology* **41**, 245–256.

3 KATAN J., GRINSTEIN A., GREENBERGER A., YARDEN O. & DeVAY J.E. (1987) The first decade (1976–1986) of soil solarization (solar heating): a chronological bibliography. Phytoparasitica **15**, 229–255.

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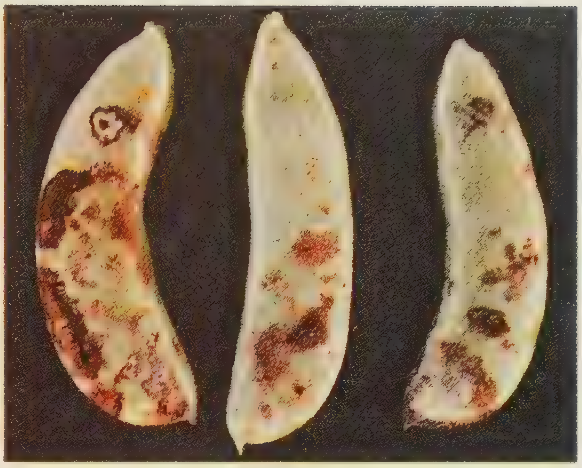
SOIL ROT of beans caused by
***Thanatephorus cucumeris* (Frank) Donk**
Sclerotial state: *Rhizoctonia solani* Kühn

OCCURRENCE This disease was first described on common beans and lima beans in the southern USA (6). It also occurs in the north (1). Other records include Puerto Rico, Nicaragua, El Salvador, Costa Rica (2,3), Panama, Venezuela, Brazil, Argentina (5), the Philippines, India, Sri Lanka and Japan.

SYMPTOMS Lesions on pods are circular or irregular, markedly sunken, and reddish brown with a dark border (1,5,6), rather similar in the early stages to those of anthracnose (q.v.). Under humid conditions, however, there is profuse development of wefts of mould which bind the pods together (6). Seeds may be invaded (1,5).

BIOLOGY This fungus persists in the form of resting bodies (sclerotia of 0.3 to 0.5 mm) and may also produce the sexual stage (a hymenium giving rise to basidiospores). Both states are common in crop debris and on weeds (2,6). Furthermore, mould may be deep-seated in seed at the time of sowing (5). Cool moist conditions tend to favour seedling blight, basal stem rot and root rot, which are the characteristic symptoms in temperate growing areas (4). Pods in contact with the ground may be invaded (1). In the humid tropics, however, the fungus is also able to attack the aerial parts of mature bean plants, causing the field disease known as 'web blight' (from the mould strands which envelop leaves, stems and pods) (1,3,5,6). Initial infection may be by airborne spores causing leaf spots which expand rapidly in warm wet weather (2). Alternatively, the main source of infection may be the minute sclerotia and mould fragments which are dispersed by wind, rain, irrigation water, animals and farm machinery (5,6); one study has demonstrated the particular importance of soil splash (3).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At temperatures between 25° and 30°C disease development is very rapid if moisture is present (3). The same may be said of post-harvest decay, 'nests' of mould developing in apparently sound produce within a day or two of picking (6).

CONTROL Infested crop debris should be promptly removed and destroyed; it may also be beneficial to drench the soil with a fungicide (3,4) or to use the method of soil solarization (page 132). Clean or treated seed should be sown (4,5). Intercropping with maize has been recommended, as this crop is not susceptible to the disease. The use of a mulch is advantageous in preventing soil splash (3), and fungicide sprays give added protection. Harvested pods should be carefully sorted and kept dry; rapid cooling is desirable since the fungus grows very slowly at temperatures below 10°C (6). Much effort has gone into the breeding of bean cultivars with increased resistance to this disease.

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2 ECHANDI E. (1965) Basidiospore infection by *Pellicularia filamentosa* (=*Corticium microsclerotia*), the incitant of web blight of common bean. *Phytopathology* **55**, 698–699 illus.

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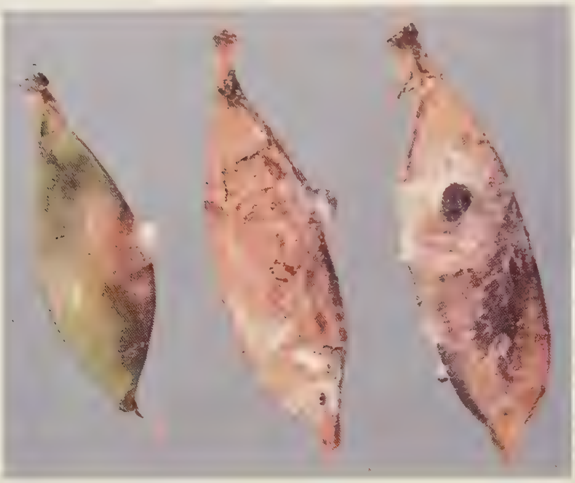
WHITE MOULD and WATERY SOFT ROT of peas and beans caused by *Sclerotinia* spp.

164



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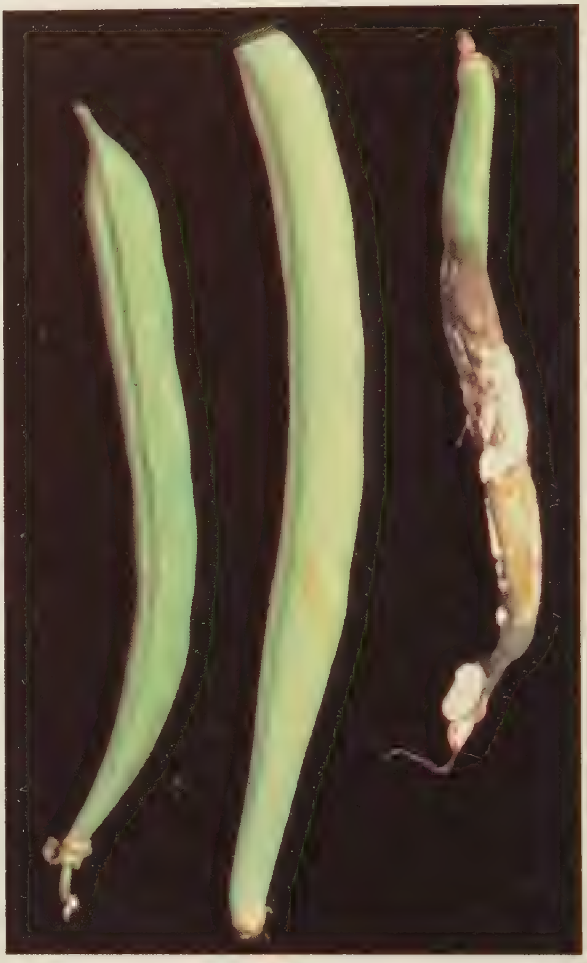
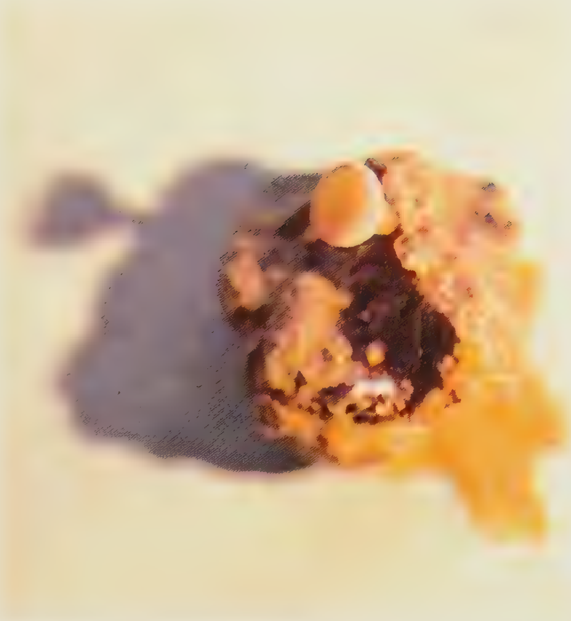


OCCURRENCE White mould is one of the most important diseases of common beans in, for example, the USA (6,7), Canada (1,9), Italy (4) and Australia (10). The disease may be caused by *Sclerotinia sclerotiorum* (Lib.) de Bary (8), *S. minor* (page 212) (4) or both (10). The same fungi have been found on peas; *S. sclerotiorum* appears to predominate in the USA, Argentina, India, New Zealand and the UK (2), and *S. minor* in southern Italy (4). *S. sclerotiorum* has been recorded on lima beans in the USA. Records on faba beans include *S. sclerotiorum* in Switzerland, *S. minor* in Israel, *S. trifoliorum* Eriksson in Sweden and *S. trifoliorum* var. *fabae* Keay in the UK (3).

SYMPTOMS Lesions on pods are at first watersoaked, later brownish, becoming covered with a dense white mould (10). Tough resting bodies (sclerotia) develop; as they mature they exude drops of liquid and gradually change from white to black (or, in *S. trifoliorum* var. *fabae*, to brown). Sclerotia are variable in size, but those of *S. minor* tend to be between 0.5 and 2 mm across (and are often coalescent), those of *S. sclerotiorum* are generally 2 to 15 mm, and of *S. trifoliorum* 2 to 20 mm.

BIOLOGY These fungi may be transmitted to new areas as sclerotia in (or mixed with) seed (9). Sclerotia also persist on crop debris and in the soil (2), and the various species have contrasting modes of development. In wet conditions the small sclerotia of *S. minor* characteristically give rise to a dense mass of mould strands which extend along or just below the soil surface and attack peas and beans at the stem-base (4,10). Plants may wilt and die before any pods are produced; infected stems become brown, dry and hollow, later filled with sclerotia (4). Occasionally *S. minor* may invade pods in contact with the ground (10). The sclerotia of the other species require a wet period lasting several weeks, after which they produce the sexual stage (apothecia giving rise to ascospores) (2,7). Temperature also is probably influential; in the UK *S. sclerotiorum* tends to produce apothecia in spring and summer, while *S. trifoliorum* generally produces most during autumn and winter. Even if rain or irrigation is infrequent, the microclimate beneath bushy maturing plants is often sufficiently cool and moist to favour disease development (7). Spores are discharged into the air and, although they generally travel only a few metres (8), air currents may carry them into or from adjacent fields (1). Infection from spores occurs only via moribund tissue; thus pea and bean crops become susceptible during flowering, when some of the blossoms begin to wither (5). If plants stay wet for two or three days the fungi invade dying flowers which may remain attached to developing pods or fall and adhere to plant parts (7). It may be seen that the mode of infection is essentially different from that of *S. minor* (which is capable of producing apothecia and ascospores in the laboratory but rarely does so in nature). All these fungi can cause heavy losses in the field (1,2,4,5,7,9). In addition, pods appearing healthy at harvest time may rot during transit or storage (6), 'nests' of decay developing most rapidly at temperatures between 20° and 25°C (4).

CONTROL Depending on location of the growing area, sclerotia in the soil may be killed by a period of flooding or by solarization (page 132). There have been numerous attempts at biological control, in which certain naturally occurring organisms are added to the soil to parasitise sclerotia, either before or after the crop is planted. Much work has been done on ways of modifying cultural practices, the aim being to minimise the length of time the bean plants remain wet (from rain, irrigation water or dew) (7). Disease incidence is reduced by wide spacing and careful orientation of rows so as to take advantage of prevailing wind and sun to facilitate ventilation and drying of foliage (10). For the same reason, the less bushy (including semi-leafless) cultivars of peas and beans are more likely to escape disease (7). Control of weeds is important; besides the fact that many can act as alternative hosts of *Sclerotinia* spp. (2), their presence restricts air movement through the crop (1,10). Systemic fungicides can be effective, provided that spraying commences before full bloom and that there is good coverage of blossoms (10). In addition, a post-harvest fungicide dip may be used (6). Refrigeration retards but does not halt the development of mould (4).



166 Soil sample from bean field, showing single apothecium arising from buried sclerotium

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1 BOLAND G.J. & HALL R. (1988) Numbers and distribution of apothecia of *Sclerotinia sclerotiorum* in relation to white mold of white bean (*Phaseolus vulgaris*). *Canadian Journal of Botany* **66**, 247–252.
2 JONES D. & GRAY E.G. (1973) Factors affecting germination of sclerotia of *Sclerotinia sclerotiorum* from peas. *Transactions of the British Mycological Society* **60**, 495–500 illus.
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10 WONG J.A., COX J.I. & MAYNARD J.R. (1980) White mould in green beans: research progress. *Journal of Agriculture, Tasmania* **51**, 108–111 illus.

See also:

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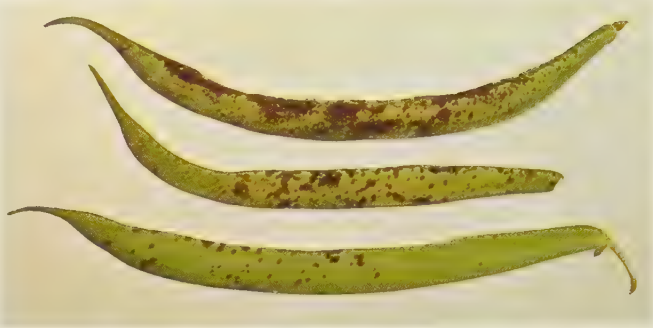
PHILLIPS A.J.L. (1989) Fungi associated with sclerotia of *Sclerotinia sclerotiorum* in South Africa and their effects on the pathogen. *Phytophylactica* **21**, 135–139.

OTHER DISEASES of peas and beans



167 Brown spot of beans

BACTERIAL SOFT ROT may be caused by *Erwinia carotovora* ssp. *carotovora* (page 271), which an attack tissue weakened by chilling, freezing, sunscald or bacterial blight (q.v.). In a warm moist storage environment pods become soft, slimy and evil-smelling. A mixed rot has been reported on peas in Egypt (3) caused by the bacterium *E. herbicola* var. *herbicola* and the fungus *Geotrichum candidum*. Control is by careful handling and rapid cooling of harvested pods.

BROWN SPOT of common beans, caused by *Pleiochaeta setosa* (Kirchn.) Hughes, should not be confused with bacterial brown spot (q.v.). The fungal brown spot disease has been recorded in Australia (7), and is characterised by slightly sunken spots with dark brown to black centres and paler margins. The lesions, which may be anywhere on the pods, gradually enlarge and coalesce, reducing the market value of the pods. No spores are found on the lesions. The fungus survives on weeds and can attack beans only if they have been damaged, for example by sand particles splashed up by wind-blown rain (8).

CHOANEPHORA ROT of peas and beans has been reported from the southern USA after a spell of hot wet weather (5). The disease has also been recorded on peas in Japan. The fungus, *Choanephora cucurbitarum* (page 27) characteristically invades dying blossoms and subsequently causes a soft wet rot, with the production of quantities of spores (conidia). Control is by spraying the crop at flowering time, and ensuring prompt cooling of harvested pods.

CLADOSPORIUM ROT of peas is caused by *Cladosporium pisicolum* Snyder, and has been recorded in the western USA and in India. Symptoms consist of dark brown to black sunken scabs of irregular shape, bounded by well-defined raised black margins (8). The fungus survives in crop debris, and also in seed. Disease development is favoured by cool foggy weather, when wind-dispersed spores (conidia) are able to infect dying blossoms still attached to developing pods. *C. cladosporioides* (page 64) has been recorded in Argentina, causing a marked blemish on pea pods and seeds. *C. herbarum* (page 64) causes spotting of shelled green lima beans. Control of these fungi is by crop rotation, the avoidance of low-lying damp terrain, and the use of clean seed.

FUSARIUM ROT, caused by *Fusarium pallidoroseum* (page 34), has been recorded as a market disease of pea pods in India, and also as a serious field disease of common beans in Brazil, following prolonged wet weather (1). Lesions are at first watersoaked and later brown; if humid conditions continue there is profuse development of mould. The fungus is transmitted in seed, and can cause rotting of all parts of the plant.

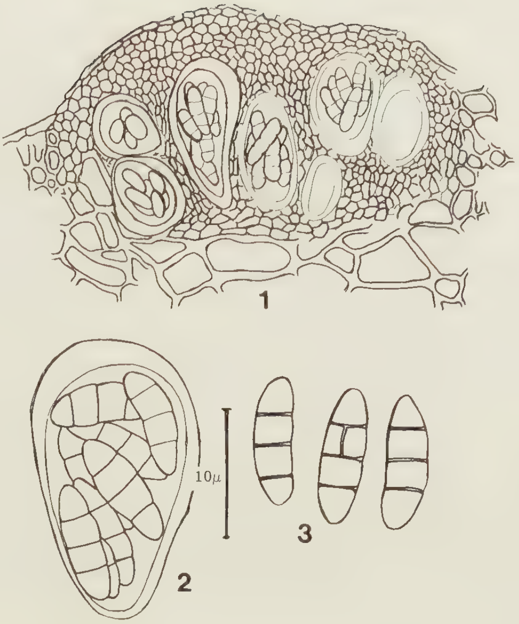
HELMINTHOSPORIUM SPOT of common beans has been observed in the southern USA (10), in a crop which had been grown near to oats infected with *Helminthosporium victoriae* Meehan & Murphy. Lesions on bean pods were small watersoaked spots (about 1 mm across) with black centres and indefinite margins; high humidity induced sparse production of spores (conidia).

PEA SEED-BORNE MOSAIC is caused by a virus, reported from Japan, the USA and parts of Europe. Pods from infected plants tend to be undersized, have line patterns, and contain either no seeds or one or two seeds with split coats (as in downy mildew, q.v.). Recommended measures include the use of virus-free seed and the control of aphids which transmit the virus from plant to plant.

PINK MOULD ROT, caused by *Trichothecium roseum* (page 41), has been recorded on stored green pea pods in India, and on common beans in Canada (9). Lesions are at first watersoaked, later chocolate-brown. Humid conditions permit the development of a powdery mould which initially appears white but eventually assumes a salmon-pink colour. Infected seeds become yellowish and shrivelled, later mouldy. The fungus is a common soil inhabitant and its spores are dispersed by air currents. Disease development is associated with dense plantings which result in prolonged retention of dew and raindrops. *T. roseum* has also been found growing on lesions of *Uromyces viciae-fabae* (faba bean rust, q.v.).

SCAB of lima beans is caused by the fungus *Elsinoë phaseoli* Jenkins. The disease appears to be of particular importance in Central America and the Caribbean, and records include Nicaragua, El Salvador, Mexico, Puerto Rico and Cuba (CMI Map 194). Lesions, which are sometimes especially abundant on one side of the pod, are characteristically swollen and brightly coloured, brick-red to brown when young, subsequently light brown bordered with dark brown or reddish-purple (4). Both stages of the fungus may develop on the lesions, that is the sexual state (pseudothecia giving rise to ascospores) and the asexual state (acervuli and conidia of the *Sphaceloma* type). Recommended control measures include crop rotation, sanitation and seed treatment.

SEPTORIA BLIGHT or **BLOTCH** of peas is caused by *Septoria pisi* Westend., which has been recorded in, for example, the USA and New Zealand (2). Pods may be infected, the pale brown lesions being dotted with minute black bodies (pycnidia giving rise to conidia) which survive the winter in crop debris. Spores are dispersed by watersplash and wind-blown rain and, at moderate temperatures and high relative humidity, can cause infection of leaves and pods. Control is by crop rotation. Some pea cultivars are partially resistant to the disease.



168 *Elsinoë phaseoli* (from CMI Descr. No. 314, Sivanesan & Holliday 1971) 1 pseudothecia 2 ascus 3 ascospores

YEAST SPOT may be caused by *Nematospora coryli* Peglion or *N. gossypii* Ashby & Nowell (CMI Descr. 184, 185). The former has been recorded on lima beans in the USA, while both species occur on common beans in Brazil (6). Pods which are apparently healthy may contain badly diseased seeds, showing dark brown sunken lesions on which may be found the yeast's sexual stage (asci giving rise to ascospores). The optimal growth temperature for these organisms is approximately 30°C.

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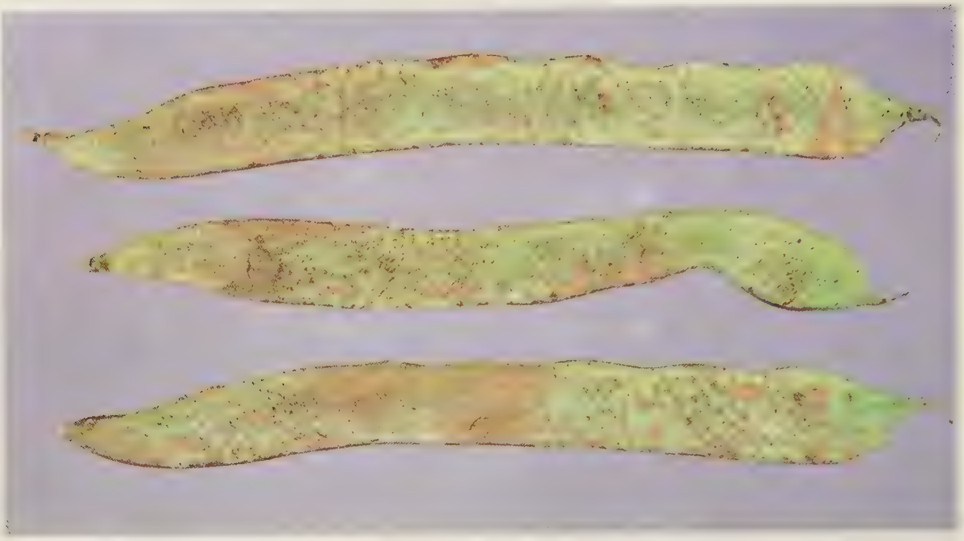

169 Chilling injury of phaseolus beans

CHILLING INJURY of bean pods takes the form of surface pitting, diagonal brown streaks (loosely termed 'russeting'), a general dullness, and increased susceptibility to decay. Injury may be induced within a few days at temperatures below 5°C, although cultivars differ markedly in sensitivity. Furthermore, symptoms may not be apparent until the beans have been returned to ambient temperature for a day or so (2,5). The presence of free moisture aggravates the effects of chilling. Recommended storage temperatures for fresh bean pods are usually within the range of 5° to 8°C, in order to minimise the risk of chilling injury. Higher temperatures promote yellowing (q.v.).

PHYSICAL INJURY is often characterised by the presence of dull brown marks, and in beans may be difficult to distinguish from chilling injury. Damage can result from wind-blown sand, machine-harvesting or rough handling of the pods.

SUNSCALD is indicated by brown or reddish spots which are typically on one side of the pod only (3). The spots may develop into streaks, which eventually coalesce, becoming watersoaked and slightly sunken. Symptoms are evident at harvest time, and affected pods should be culled at source.

WILTING is especially deleterious in mangetout peas and fine green beans. Moisture loss can be minimised by appropriate plastic film packaging, coupled with prompt reduction of temperature (4).

YELLOWING and **TOUGHENING** are aspects of senescence, which can be retarded by proper temperature management, the application of a growth-regulating chemical (1), and the elimination of ethylene from the storage environment. Even under optimal conditions, however, storage life of pea and bean pods is limited to a week or so.

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CHAPTER 5

BRASSICAS

The 'cole crops' or 'brassicas' belong to the family Cruciferae (7). The genus *Brassica* originated in the Old World; most cultivars thrive only in temperate regions though some are also important in the tropics. The present chapter is concerned with those species which are grown for their leaves, buds or inflorescences; those grown for their 'roots' (swedes and turnips) are considered in Chapter 8.

The **cabbage**, *Brassica oleracea* var. *capitata* L., is characterised by the formation of a single, large 'head' (composed of overlapping leaves) which may be loose or compact. Its ancestors were non-heading types, similar to **kale**, *Brassica oleracea* var. *acephala* DC., which has been cultivated in Europe for thousands of years. Modern cabbage cultivars include savoys with crinkled green leaves, red cabbages used mainly for pickling, and winter white cabbages which can be eaten fresh or cooked or made into 'coleslaw'. Some cultivars of white cabbage have been bred specially for pickling into sauerkraut. Chinese cabbage (q.v.) is a different species.

The **cauliflower**, *Brassica oleracea* var. *botrytis* L., evolved in the eastern Mediterranean region and was first described in the Middle Ages. It is characterised by a dense 'curd' which is an inflorescence of aborted flowers. Sometimes the leaves are tied over the developing curd to maintain its pure white colour.

Broccoli can mean 'heading broccoli', a hardy type of cauliflower with a yellowish or greenish curd. More usually, however, it refers to sprouting broccoli, *Brassica oleracea* var. *italica* Plenck, in which there are clusters of true flower buds, green or purple in colour. In some cultivars the stem is loosely branched, and numerous small inflorescences are interspersed with shoots and leaves. In others the stalks are thick and sturdy, and support large masses of flower buds. This latter type of sprouting broccoli is known as calabrese (after Calabria in Italy). Since being introduced into the USA by Italian immigrants, it has become popular on both sides of the Atlantic (7).

Brussels sprouts, *Brassica oleracea* var. *gemmifera* DC., were not reliably described until the beginning of the 19th century; they first received attention in Belgium and northern France. Production is confined to cool temperate regions, and the crop is especially important in the UK (3). The 'sprouts' or 'buttons' are axillary buds which develop on the single stem; they resemble miniature cabbages, about 3 cm in diameter. A substantial proportion of the crop (especially in the USA) goes for freezing.

The **Chinese cabbage** is *Brassica campestris* ssp. *pekinensis* (Lour.) Olsson. In China it is called pe-tsai, and in Europe it is sometimes known as Chinese leaves. The heads may be rounded or cylindrical, and are composed of densely packed pale green leaves. A more primitive, non-heading type is pak choy, *Brassica campestris* ssp. *chinensis* (Rupr.) Olsson. Both are of great importance in the Far East (4), and have recently become popular in the USA and Europe (1,6).

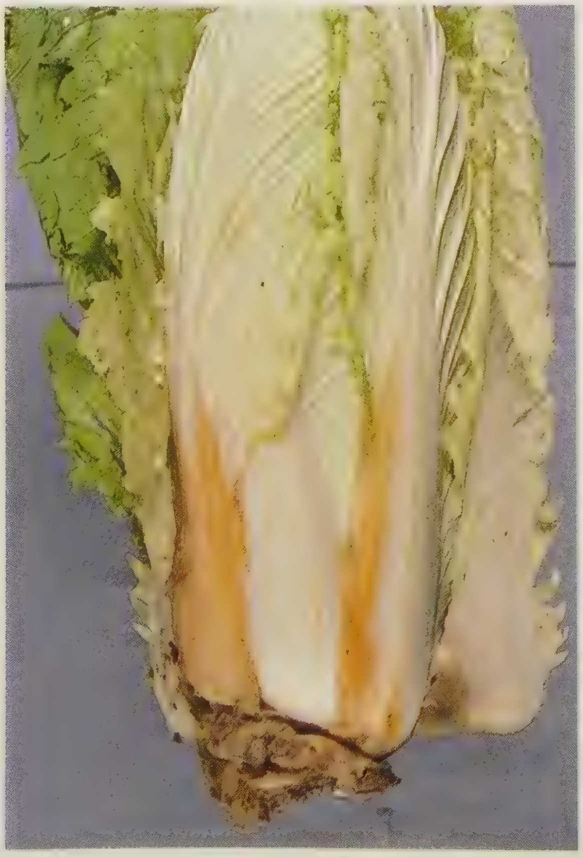
Production figures for cabbages sometimes include Chinese cabbages, broccoli, Brussels sprouts and kale. The world total is of the order of 40 million tonnes, the main producers being the USSR, China, Japan, South Korea, Poland, the USA, Romania, the UK, Turkey and Yugoslavia. Data for cauliflowers

170 Sprouting broccoli

often include 'heading broccoli'. World production is about 5 million tonnes, produced mainly in China, India, France, Italy, the UK, the USA, Poland, Spain, Japan and East Germany.

The various cole crops differ significantly in their post-harvest characteristics, reflecting intrinsic differences in structure and physiological function of the edible part (7). The fresh young leaves of kale have a very high respiration rate and, with their large surface area, are prone to wilting. Vacuum cooling or hydro-cooling is appropriate, and maximum storage life is about 3 weeks. Plastic film packaging helps to maintain quality during the brief marketing period.

Mature cabbages have a low to moderate respiration rate, and can be stored for several months (3). Maintenance of quality (including prevention of decay) depends on retardation of senescence, and it is therefore important to prevent an accumulation of ethylene. This entails care in handling, so as to minimise wounding, together with hygienic practices such as the proper disposal of trimmings. Also necessary is complete segregation from commodities such as apples which are potent producers of ethylene. Ventilated barn storage can be adequate during winter, but refrigeration is necessary for prolonged storage. It is desirable to keep the temperature as low as possible without risk of freezing; thus, for most practical purposes, the recommended storage temperature is 0°C. This maintains a very low respiratory activity in the cabbage and greatly inhibits (though does not completely suppress) the growth of grey mould, the most important disease organism affecting cabbages (3,8,9). With regard to relative humidity, the relationships between host, disease organism and environment are complex, and recommendations differ as to the optimal compromise. Some research workers have found that, coupled with low temperature, a relative humidity close to saturation point (98 to 100%) is beneficial in extending storage life, turgid tissue being resistant to fungal invasion. Other workers have found that such high humidities increase fungal spoilage (conceivably because slight wilting, and hence predisposition to infection, has occurred between field and store). They advocate a slightly lower relative humidity (approximately 95%), which results in significant dehydration of the outermost leaves, to the extent of precluding mould growth. The dried wrapper leaves act (like onion 'skins', q.v.) as a barrier, protecting the succulent inner tissues from moisture loss and infection (3).

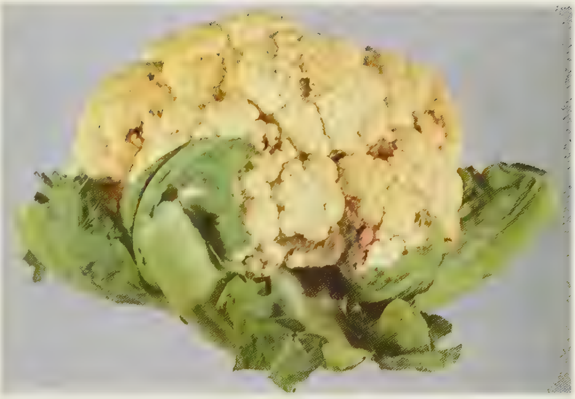

171 Bacterial soft rot of Chinese cabbage

Fungal rotting can be further controlled by the application of an appropriate fungicide immediately before storage. Dusts, fogs and sprays have been used, but dips and drenches are more common (3). When cabbages are treated with a liquid fungicide, thorough draining is essential before storage, since excessive moisture retained between the leaves is likely to induce bacterial soft rot (q.v.). Storage life of winter white cabbage can be significantly extended by the use of controlled atmospheres, the primary effect being retardation of senescence. Some cultivars can be held for up to 10 months, continuity of supply being especially important to processors (3).

Cauliflowers benefit from rapid cooling and refrigerated storage, but they have a limited storage life compared with cabbages. Even under optimal conditions (0°C and 95% relative humidity) cauliflowers can be kept for a few weeks only, the main problem being deterioration of the delicate curd tissue. Gentle handling is essential, since physical damage permits infection by fungi and bacteria. Prolonged exposure to light can cause the curd to take on a yellowish hue. Signs of senescence include yellowing and abscission of the wrapper leaf stalks, together with 'riciness' and discoloration of the curd. Controlled atmosphere storage appears to confer little advantage; indeed, it may cause deleterious effects which become apparent only after the cauliflower is cooked (6). If plastic film packaging is used, the film should be perforated in order to prevent suffocation and decay.

Sprouting broccoli is renowned for its extremely high rate of respiration, consequent upon the great physiological activity shown by the fast-growing flower buds. Weight for weight, broccoli produces ten times as much heat as cabbages. These properties are reflected in the fact that broccoli has a very short storage life, only a few days at ambient temperature and generally not more than 2 to 3 weeks at 0°C and 95% relative humidity. Rapid temperature reduction is essential, whether it be by forced-air cooling, hydro-cooling or by the use of crushed ice in the packages. Inadequate pre-cooling can lead to self-heating of broccoli whilst in transit. In warm conditions senescence is rapid; the flower buds start to open and the fresh green colour gives way to yellow. Ethylene produced by the fading tissues (or from other sources) has in turn a deleterious effect on nearby fresh broccoli.

Controlled atmosphere storage can be effective in delaying the onset of senescence, though care is necessary to prevent undesirable changes in odour, flavour and texture (6). Plastic film packaging can be used to to provide a modified atmosphere but, depending on the film, it may be necessary to perforate it when the broccoli is moved from cold store to market (10).


172 Grey mould rot of cauliflower

Brussels sprouts are best stored 'on the stem', and in this way can be held in store for several weeks at 0°C and high humidity (3). At higher temperatures (5°C and above) a controlled atmosphere can be beneficial, although there have been some reports of internal discoloration, bitterness and off-odours (5,6). Sprouts have a fairly high rate of respiration. Once removed from the stem, they become increasingly prone to deterioration, including discoloration of the cut surface, opening of the leafy buds, yellowing and decay (3). The deleterious effect of ethylene must be mentioned once again (5).

Chinese cabbages have been successfully stored at 0°C and 95% relative humidity for at least 2 months. There are reports of chilling injury in some cultivars, however, and alternative recommendations are a storage temperature between 2.5° and 5°C, coupled with a controlled atmosphere regime (1).

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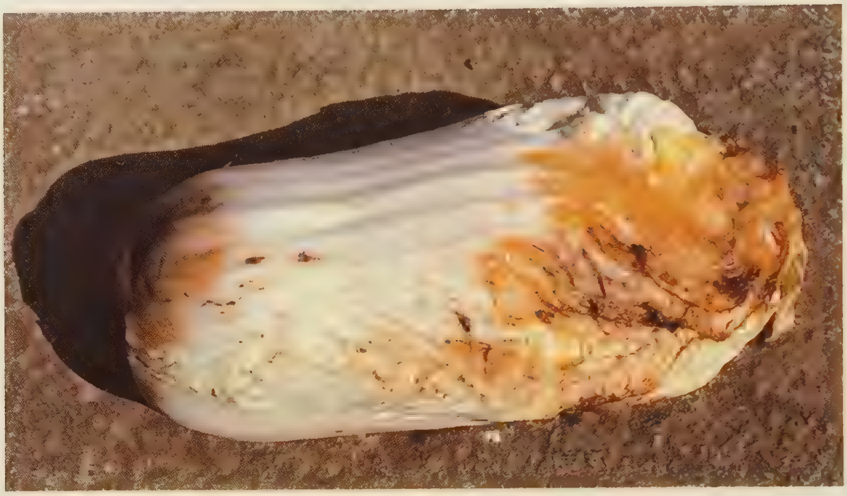
BACTERIAL ROTS of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages

173



Bacillus polymyxa **(Prazmowski) Mace** and ***B. pumilus*** **Meyer & Gottheil** are reported to cause a head rot of cabbage, decay being especially severe at temperatures between 25° to 30°C (5).

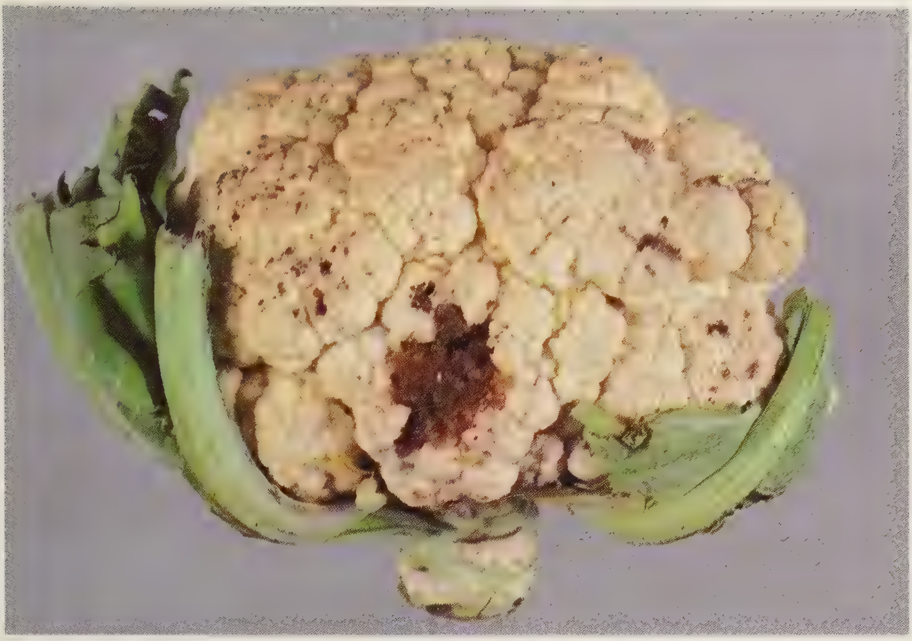
Erwinia carotovora **ssp. *carotovora*** and ***E. carotovora*** **ssp. *atroseptica*** (page 302) each causes a bacterial soft rot. Watersoaked lesions appear, the tissue becomes extremely soft, and a slimy decay ensues. Secondary bacteria impart a disagreeable odour to decayed tissue. Rotting may be initiated in the field, after bacteria from the soil have invaded the outermost leaves and the stem; hot humid weather is particularly conducive to infection (3,4,6,7). Insects may be responsible both for causing injury to the plant and for carrying the bacteria (4). Infection can also occur during harvesting and handling, through the cut stem or via wounds, cauliflower curds being especially vulnerable. Infected tissue is stimulated to produce ethylene, a further deleterious effect of bacterial attack. Control measures include strict field hygiene, and restriction of fertiliser so as to maintain tissue resistance. Copper sprays can give some protection (3), and control of insects may also be necessary (7). Care should be taken to avoid injuring produce during harvesting and handling, and to keep it cool after harvest. Some resistant cultivars are available (4,7).

Pseudomonas cichorii **(Swingle) Stapp** causes bacterial spot of cabbage, cauliflower, broccoli, Brussels sprouts and Chinese cabbage (8). In one report the bacterium is said to be capable of infecting uninjured cabbage leaves, inducing slightly sunken brownish lesions. Another report sates that wounds are a prerequisite for infection, and that lesions on cabbages have a characteristic zoned appearance, hence the name 'bacterial zonate spot' (8). Infected cabbage tissue remains firm unless invaded by secondary soft rot bacteria. Lesions on the other hosts lack zonation and tend to be soft but not slimy (8). The optimal temperature for decay is 26°C and the bacterium thrives in conditions of poor ventilation (8). It makes slow growth at 5°C.

Pseudomonas syringae **pv. *maculicola*** **(McCulloch) Young et al.** also causes a bacterial spot, and has been recorded on cabbage, cauliflower and Brussels sprouts (1). The spots are at first watersoaked and circular, later brownish-grey with dark borders, irregularly angular and coalescent. The disease occurs in wet seasons, and infection appears to take place via the natural openings (stomata) in the leaves. Suggested control measures include crop rotation and the use of healthy seed (1).

Pseudomonas marginalis **pv. *marginalis*** **(Brown) Stevens** causes a slimy soft rot, and has probably often been confused with the erwinias (q.v.), from which it differs in its ability to cause decay at temperatures as low as 0°C (2). Infected tissue is watersoaked and discoloured, and after cool moist weather there may be severe losses in both field and store (10). Frost damage also predisposes crops to infection.

Pseudomonas viridiflava **(Burkh.) Dowson** causes a soft rot, for example in cauliflower (9). Irregular dark green watersoaked lesions develop, usually at the leaf margins. In advanced stages the tissue becomes brown, soft and watery, and may have a putrid odour. Bacteria are distributed through



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the leaf in the conductive tissue (vascular bundles), resulting in systemic infection (9). Cold wet weather predisposes plants to infection. If dry conditions ensue, the lesions become papery.

Xanthomonas campestris **pv.** ***campestris*** causes black rot (q.v.), which is essentially a field disease but which predisposes crops to infection by secondary soft rot bacteria.

Since all these bacteria require moisture for infection, it is important to ensure that harvested crops are sufficiently dry to withstand long-term storage. The use of post-harvest fungicide dips and drenches for cabbages results in retention of liquid between the leaves, and this should be removed by efficient ventilation at the start of the storage period. Cultivars differ in susceptibility to bacterial rots.

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See also:

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BLACK ROT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Xanthomonas campestris **pv.** ***campestris*** **(Pammel) Dowson**

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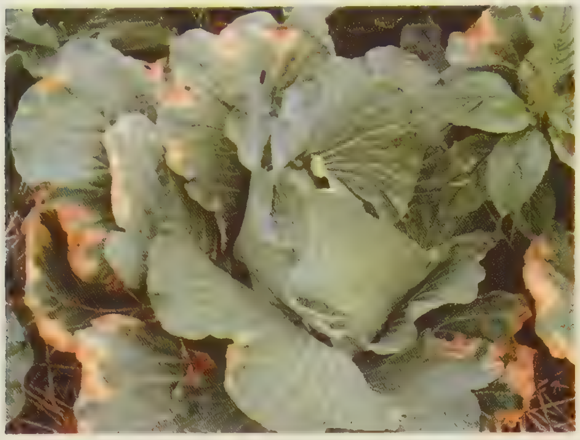


OCCURRENCE This bacterial disease, worldwide in distribution, is especially important in tropical, subtropical and humid continental regions (5). Records include India (3), Taiwan, East Africa (4), South Africa, Brazil, Hawaii (1) and the USA (2,5).

SYMPTOMS Yellowish-brown V-shaped lesions develop at the leaf margins, and the veins turn black (5). If the stem is cut transversely a ring of black 'dots' may be visible. Affected tissue is firm at first, but may subsequently be invaded by secondary bacteria, leading to a soft rot (5).

BIOLOGY The bacteria survive on crop debris in the soil (1) and may be introduced into new areas on contaminated seed (2). From the soil the bacteria are able to invade roots, especially if these have been injured, for example by insects (5). Contaminated seeds give rise to infected seedlings which may, however, be symptomless (5). Bacteria are distributed throughout the plant in the conductive tissue (vascular bundles), this being known as systemic infection. When liquid exudes through natural openings at the leaf margins (guttation through the hydathodes), the bacteria are expelled also, and are later carried to healthy plants by wind-blown rain, field workers or farm machinery. When liquid is drawn into the leaves, new infections may occur (4). Epidemic development is favoured by warm wet conditions. Harvested produce is marred by disfiguring blemishes, and is predisposed to invasion by secondary soft-rot bacteria (5).

CONTROL Crop rotation with non-cruciferous crops is important. It may be necessary to disinfest the seed-bed, and it is advantageous to sow seed which has been produced in cool dry regions where black rot is uncommon (5). Chemical treatment of seed is preferable to a hot-water soak (2). Seed should be tested and certified free of the black rot organism, since even a very low level of contamination can result in significant disease in the growing crop (2). Certification should be extended to transplants shipped to new areas (5). Cultural practices influence disease development; epidemic spread is facilitated by overhead sprinkling or by field workers moving through the crop when the leaves are moist. A mulch is beneficial in that it reduces soil splash (4), thus minimising infection by way of contaminated soil particles. If a crop is found to be infected at harvest time, the plants should be carefully trimmed and marketed without undue delay. Field debris should be destroyed. Some cultivars are resistant to black rot, and breeding work continues (3).

---

1 ALVAREZ A.M. & LOU K. (1985) Rapid identification of *Xanthomonas campestris* pv. *campestris* by ELISA. *Plant Disease* **69**, 1082–1086.

2 HARMAN G.E., NORTON J.M., STASZ T.E. & HUMAYDAN H.S. (1987) Nyolate seed treatment of *Brassica* spp. to eradicate or reduce black rot caused by *Xanthomonas campestris* pv. *campestris*. *Plant Disease* **71**, 27–30.

3 JAMWAL R.S. & SHARMA P.P. (1986) Inheritance of resistance to black rot (*Xanthomonas campestris* pv. *campestris*) in cauliflower (*Brassica oleracea* var. *botrytis*). *Euphytica* **35**, 941–943.

4 ONSANDO J.M. (1987) Management of black rot of cabbage (*Xanthomonas campestris* pv. *campestris*) in Kenya. *Tropical Pest Management* **33**, 5–6.

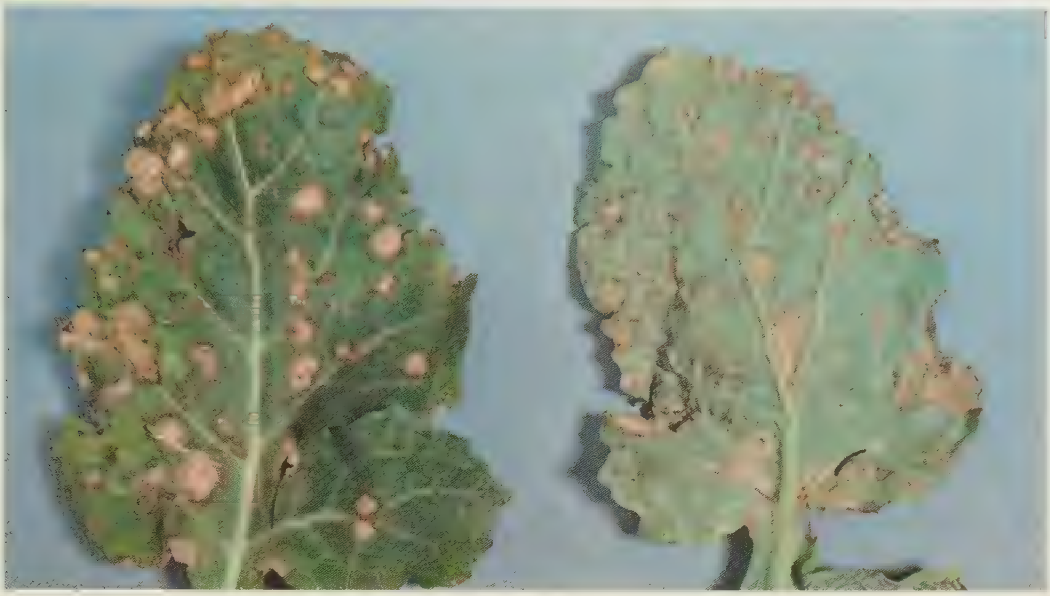
5 WILLIAMS P.H. (1980) Black rot: a continuing threat to world crucifers. *Plant Disease* **64**, 736–742 illus.

See also:

HUANG T.C. & LEE H.L. (1988) [Hot acidified zinc sulphate as seed soaking agent for the control of crucifer black rot.] *Plant Protection Bulletin, Taiwan* **30**, 245–258.

ROBESON D.J., BRETSCHNEIDER K.E. & GONELLA M.P. (1989) A hydathode inoculation technique for the simulation of natural black rot infection of cabbage by *Xanthomonas campestris* pv. *campestris*. *Annals of Applied Biology* **115**, 455–459 illus.

CERCOSPORELLA SPOT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by ***Pseudocercosporella capsellae*** **(Ell. & Ev.) Deighton**



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OCCURRENCE This disease has been recorded in several countries, including the USA (2,4), Puerto Rico (4), Canada (5), the UK, Austria (1), Germany, Algeria (3) and Korea (CMI Map 197).

SYMPTOMS Minute dark flecks appear on the outer leaves, and coalesce to give a finely branched appearance, somewhat similar to that of downy mildew (q.v.) (2,4). Subsequently, however, the affected areas became rounded spots, several mm in diameter and brownish with well-defined borders. Under humid conditions there is copious production of characteristic ash-grey spores (conidia) (4,5). The common name 'white leaf spot' is more appropriate when applied to the disease symptoms which occur on related crops such as turnips and rape (**176**) (4,5).

BIOLOGY The fungus persists in crop debris in the form of thick-walled mould strands (5), and can also survive on certain weeds (2). Cool moist weather (3) permits the production of asexual spores (conidia), which are splash-dispersed and cause infection via natural openings (stomata) in the leaves (2). In seed crops the pods may be invaded, and hence the seeds (2).

CONTROL It is desirable to practise crop rotation, bearing in mind that turnips, rape and radishes are also susceptible to the disease (2). Weeds should be controlled, and crop debris destroyed (2). It is important to sow clean seed, and to protect the developing crop with fungicide sprays. Post-harvest development of symptoms can be minimised by refrigeration, although during prolonged storage the fungus can make some progress even at temperatures below 4°C (2).

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1 BEDLAN G. (1987) Bekämpfung der Blattfleckenkrankheit an Chinakohl. *Pflanzenschutz* 3(3/4), 4–6 illus.

2 CROSSAN D.F. (1954) Cercosporella leafspot of crucifers. *Technical Bulletin of North Carolina Agricultural Experiment Station* No. 109, 23 pp illus.

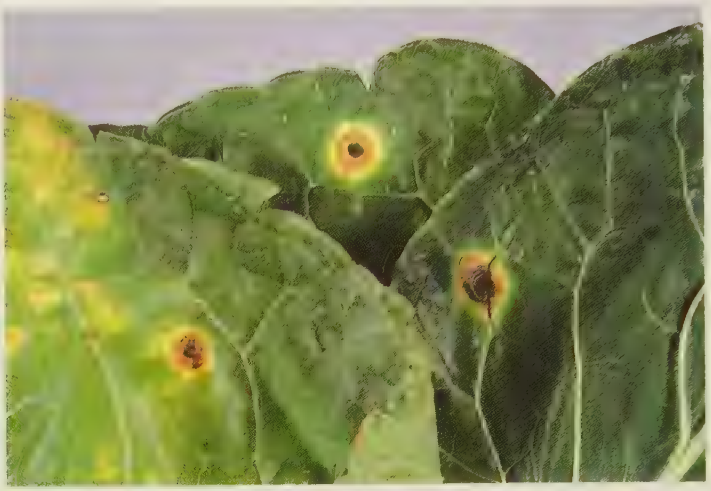
3 GUEZLANE A. & SUBRAMONIAM V. (1980) White spot disease of cabbage caused by *Pseudocercosporella capsellae*. *FAO Plant Protection Bulletin* **28**, 114–115 illus.

4 MILLER P.W. & McWHORTER F.P. (1948) A disease of cabbage and other crucifers due to *Cercosporella brassicae*. *Phytopathology* **38**, 893–898 illus.

5 PETRIE G.A. & VANTERPOOL T.C. (1978) *Pseudocercosporella capsellae*, the cause of white leaf spot and grey stem of Cruciferae in western Canada. *Canadian Plant Disease Survey* **58**, 69–72 illus.

DARK LEAF SPOT and **ALTERNARIA ROT** of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by ***Alternaria* spp.**

177



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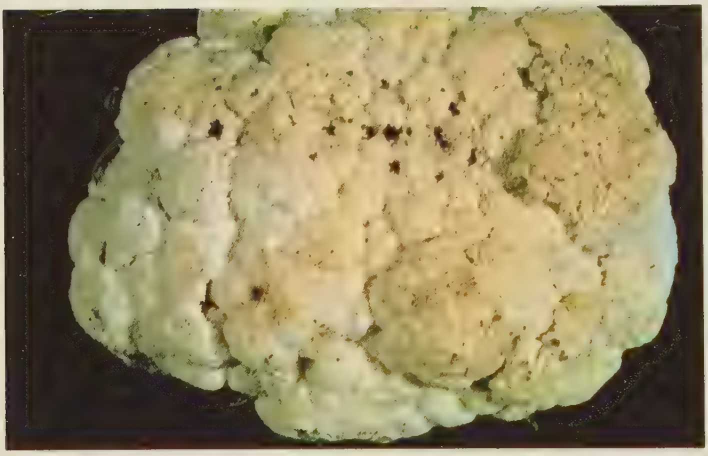
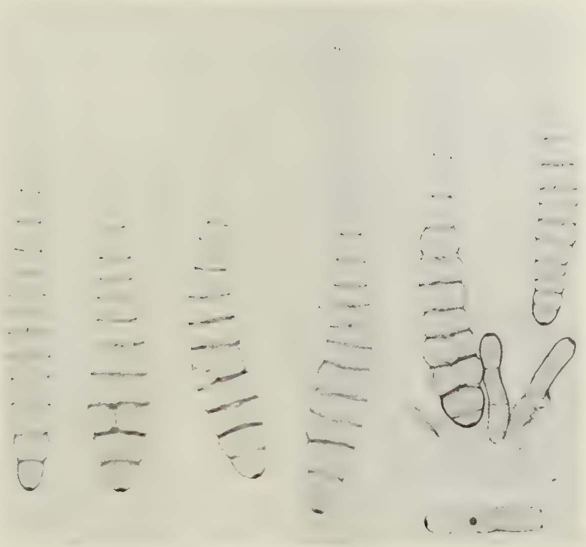


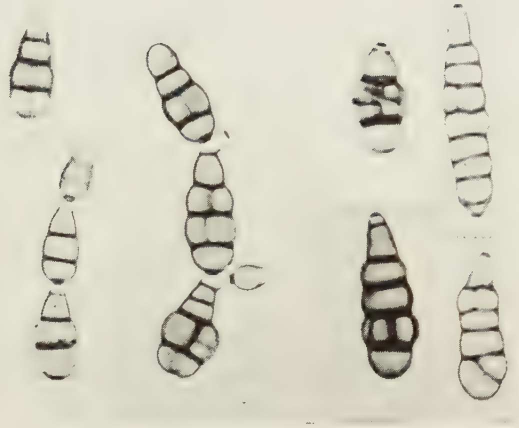
OCCURRENCE These diseases are common in many parts of the world (CMI Maps 353, 457). Records include *Alternaria alternata* (page 101) on cauliflower in East Germany (6); *A. brassicae* (Berk.) Sacc. on cabbage, kale, broccoli and Brussels sprouts in the UK (5), on cabbage and cauliflower in the USA (8) and on Chinese cabbage in the Philippines (3); *A. brassicicola* (Schwein.) Wiltshire on cabbage in France (4), the UK and Brazil (1) and on cauliflower in Finland (7) and the USA (8); and *A. raphani* Groves & Skolko on cabbage and Chinese cabbage in Japan (9) and on broccoli in the USA (2). There was much early confusion over nomenclature for the various species (9).

SYMPTOMS *A. alternata, A. brassicicola* and *A. raphani* induce very dark spots on leaves, hence the alternative common name of 'black leaf spot'; the lesions may be zonate, as in ring spot (q.v.). *A. brassicae* causes spots which are (at least initially) comparatively pale, hence the common name of 'grey leaf spot' (2,3,8). These fungi may also cause brown or black discoloration of cauliflower curds (1,8). Under humid conditions quantities of spores are produced, and microscopic examination permits identification of species (2,3,8,9,10).

BIOLOGY The fungi can persist in crop debris, and furthermore they are commonly transmitted in seed (1,5,7,10). Their spores (conidia) are dispersed by wind, and may also be disseminated by slugs (4). In wet conditions infection occurs by direct penetration of intact tissue or via natural openings (stomata) in the leaves, stems and (in seed crops) pods (6). Produce may already be disfigured at harvest and, even after trimming, can undergo serious rotting in store.



179 *Alternaria brassicae* (from CMI Descr. No. 162, Ellis 1968) Conidia and conidiophores × 500



180 *Alternaria brassicicola* (from CMI Descr. No. 163, Ellis 1968) Conidia and conidiophores × 500

CONTROL Crop rotation is advisable (3) and it should be borne in mind that related crops such as swedes, turnips and rape are also susceptible to the disease. (In the UK increased incidence of *A. brassicae* in cabbage, kale, broccoli and Brussels sprouts is believed to be the result of proximity to infected oil-seed rape) (5). In view of widespread seed infection (1,5) it is essential to treat seed before planting (7,10). Care is needed in the choice of a fungicide spray for the growing crop, since some formulations, while controlling other diseases, increase the incidence of alternaria infections. Spotted leaves should be removed at the time of harvest, and promptly destroyed. Decay progresses even at low temperatures and, for long-term storage of cabbage, it is advantageous to apply a fungicide as a post-harvest dust, spray or drench. Work is in progress to breed resistant cultivars.

---

1 BOLKAN H.A., RIBEIRO W.R.C. & DIANESE J.C. (1983) Occurrence and pathogenicity of *Alternaria brassicicola* in Brazil. *Plant Disease* **67**, 825–827 illus.

2 CHANGSRI W. & WEBER G.F. (1963) Three *Alternaria* species pathogenic on certain cultivated crucifers. *Phytopathology* **53**, 643–648 illus.

3 FAJARDO T.G. & PALO M.A. (1934) A serious leaf spot of Chinese celery cabbage, wongbok, and other cruciferous plants in Trinidad Valley, Mountain Province, Luzon. *Philippine Journal of Agriculture* **5**, 143–156 illus.

4 HASAN S. & VAGO C. (1966) Transmission of *Alternaria brassicicola* by slugs. *Plant Disease Reporter* **50**, 764–767.

5 HUMPHERSON-JONES F.M. (1985) The incidence of *Alternaria* spp. and *Leptosphaeria maculans* in commercial brassica seed in the United Kingdom. *Plant Pathology* **34**, 385–390.

6 STOLL K. (1952) Die Kohlschwärze Entstehung, Schadwirkung und Bekämpfung. *Nachrichtenblatt für den Deutschen Pflanzenschutzdienst* **6**, 81–90 illus.

7 TAHVONEN R. & AVIKAINEN H. (1987) The biological control of seed-borne *Alternaria brassicicola* of cruciferous plants with a powdery preparation of *Streptomyces* sp. *Journal of Agricultural Science in Finland* **59**, 199–208 illus.

8 WEIMER J.L. (1926) A leafspot of cruciferous plants caused by *Alternaria herculea*. *Journal of Agricultural Research* **33**, 645–650 illus.

9 WILTSHIRE S.P. (1947) Species of *Alternaria* on Brassicae. *Mycological Papers* No. 20, 15 pp illus.

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See also:

HUMPHERSON-JONES F.M. & PHELPS K. (1989) Climatic factors influencing spore production in *Alternaria brassicae* and *Alternaria brassicicola*. *Annals of Applied Biology* **114**, 449–458.

DOWNY MILDEW of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Peronospora parasitica **(Pers.:Fr.) Fr.**

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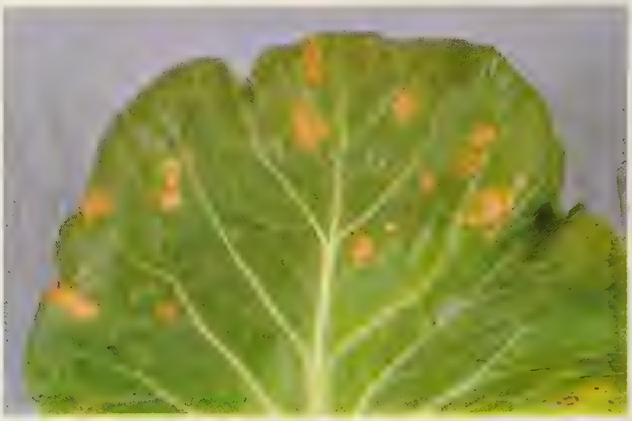

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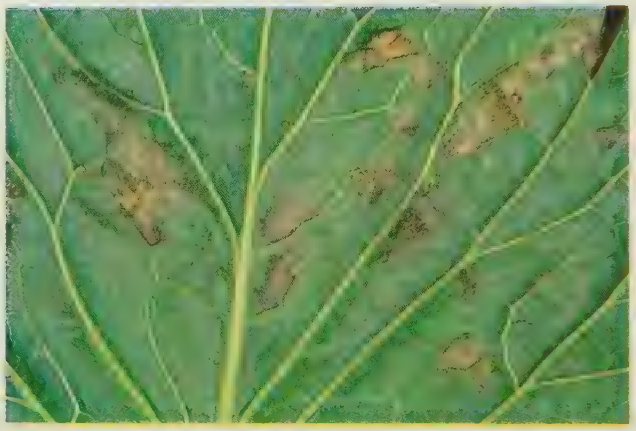
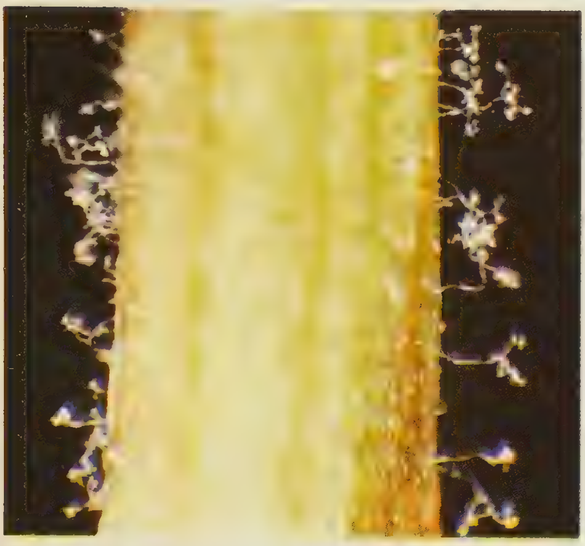


OCCURRENCE Downy mildew is widespread in temperate countries and may also occur in subtropical regions during the cool season. Records include cabbage in Canada (5) and the USA (4,9), cauliflower in the UK (8,10), Israel and northern India (1), broccoli in the USA (2) and Chinese cabbage in China (6).

SYMPTOMS The first symptoms of leaf infection are small, pale greenish-yellow spots which, being bounded by the veins, take on an angular shape. A whitish mould growth (supporting an abundance of spores) develops on the undersurface of the lesions and, in humid conditions, may also form on the upper surface. In cabbage stored for some time, lesions on leaves and stem appear greyish-black, and there may be sparse mould growth between the leaves (9). In cauliflower parts of the curd become discoloured, and the branching stems are disfigured by grey or black spots and streaks (8). Similar symptoms occur on broccoli (2). In Brussels sprouts blemishes occur on the 'buttons'. Downy mildew of crucifers is often associated with white blister (q.v.) and, during storage, is frequently followed by bacterial soft rot (8).

BIOLOGY Many races of the fungus exist, and those which attack cruciferous weeds do not usually infect crops (4). The fungus survives in crop debris in the sexual state (thick-walled oospores) and it is thought that the spores may be blown on to the developing crop during the following season and cause direct infection (7). Alternatively, or in addition, mould strands give rise to the asexual stage (sporangia/conidia) (6), spore production being profuse under cool wet conditions (4). Changes in atmospheric humidity facilitate liberation of these spores, and in the presence of free water they can cause direct infection of tissue (7). Epidemic development is favoured by temperatures between 10° and 15°C. Seedlings are especially vulnerable (4,7,10) but the fungus may also attack mature crops. Even if diseased outer leaves are removed at harvest time, incipient infections can progress in



183 Sporangiophores of *Peronospora parasitica* on cruciferous weed

184 Close-up of sporangiophores

store (8). The fungus can grow and form spores at temperatures as low as 5°C (4) and during cool storage decay can spread into adjacent healthy produce (9).

CONTROL It is undesirable to grow vegetable brassica crops in the vicinity of oil-seed rape, which is susceptible to downy mildew and can be a significant source of infection (7). In view of evidence that seed transmission may occur, it has been recommended that seed be treated with a systemic fungicide. Careful husbandry is essential; overcrowding of seedlings and excessive irrigation should be avoided (4,5). The use of chemical sprays may do more harm than good (2); some pesticides tend to dissolve away the natural waxy bloom on the leaves which, being thereby more easily wetted, are predisposed to infection. After harvest, crop residues should be promptly removed and destroyed (9). Work is in progress to breed resistant cultivars.

1 BAINS S.S., SOKHI S.S. & JHOOTY J.S. (1981) Out-break of *Peronospora parasitica* on cauliflower curds in Punjab. *Indian Phytopathology* **34**, 389–390 illus.

2 DAVISON A.D., VAUGHAN E.K. & HIKIDA H.R. (1962) Downy mildew of broccoli in the Pacific northwest. *Plant Disease Reporter* **46**, 310–311 illus.

3 DICKINSON C.H. & GREENHALGH J.R. (1977) Host range and taxonomy of *Peronospora* on crucifers. *Transactions of the British Mycological Society* **69**, 111–116.

4 HARTMANN H., SUTTON J.C. & PROCTER R. (1983) Effects of atmospheric water potentials, free water, and temperature on production and germination of sporangia in *Peronospora parasitica. Canadian Journal of Plant Pathology* **5**, 70–74.

5 HARTMANN H., SUTTON J.C. & PROCTER R. (1983) Effects of atmospheric water potentials, free water, and temperature on production and germination of sporangia in *Peronospora parasitica. Canadian Journal of Plant Pathology* **5**, 70–74.

6 HUANG K., LI G. & LIU J. (1982) [Studies on the overwintered mycelium and conidiospores of *Peronospora* in stored Chinese cabbage.] *Acta Phytopathologica Sinica* **12**(2), 63–64.

7 KLUCZEWSKI S.M. & LUCAS J.A. (1983) Host infection and oospore formation by *Peronospora parasitica* in agricultural and horticultural *Brassica* species. Transactions of the *British Mycological Society* **81**, 591–596 illus.

8 LUND B.M. & WYATT G.M. (1978) Post-harvest spoilage of cauliflower by downy mildew *Peronospora parasitica. Plant Pathology* **27**, 143–144 illus.

9 RAMSEY G.B. (1935) Peronospora in storage cabbage. *Phytopathology* **25**, 955–957 illus.

10 WHITE J.G., BLOOR K. & HO R.K. (1986) Fungicides with gamma-HCH as seed treatments for control of *Pythium* spp. and *Peronospora parasitica* on brassicas. *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108**, Supplement), 48–49.

See also:

SHERRIFF C. & LUCAS J.A. (1989) Heterothallism and homothallism in *Peronospora parasitica. Mycological Research* **92**, 311–316.

GREY MOULD ROT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Botryotinia fuckeliana **(de Bary) Whetzel**

Conidial state: *Botrytis cinerea* Pers.

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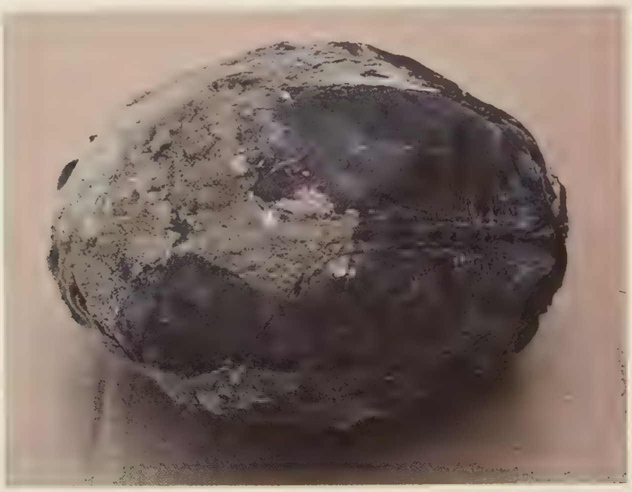

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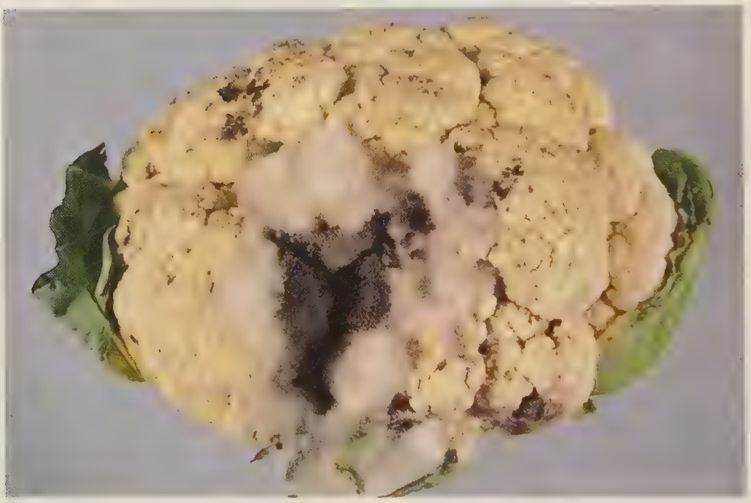
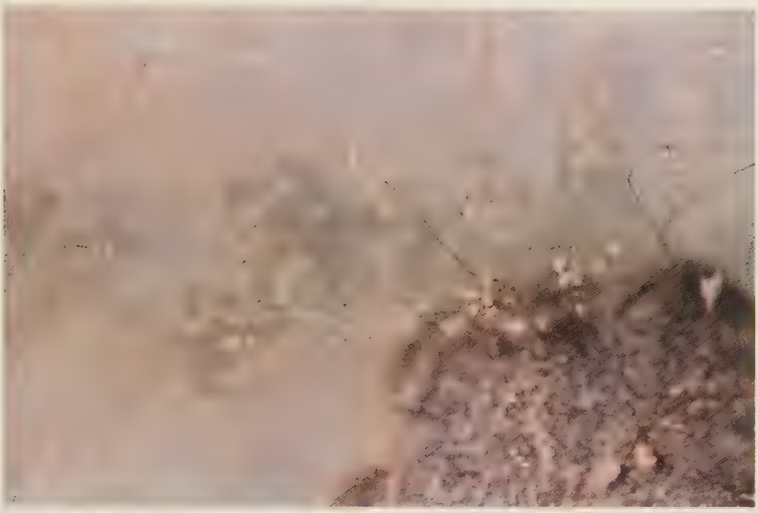


OCCURRENCE Grey mould rot is of major importance in cool temperate countries such as the UK (3,4,5), the Netherlands, Finland (9), the USSR (8), East Germany (2), Canada (7) and parts of the USA (10).

SYMPTOMS Brown watersoaked areas develop on leaf or stem tissue, and in cauliflowers and broccoli the curd may be affected. Under humid conditions a whitish mould develops, which may darken and give rise to a dense mass of brownish-grey spores.

BIOLOGY The fungus persists as resting bodies (sclerotia) in the soil or as mould strands in crop debris (10). Cool moist conditions favour the production of asexual spores (conidia) which are dispersed by wind and water-splash. During wet seasons crops may rot in the field, but the disease is usually more prevalent after harvest (10). Predisposing factors include freezing damage (whether in field or store) (4), mechanical damage due to rough handling (2,10) and prior infection by other disease organisms. Moribund leaf margins are also susceptible (7,10). Once established, grey mould subsequently progresses into sound healthy tissue, and severe ‘nesting’ may occur in store. The optimal growth temperature for the fungus is approximately 20°C (10), but significant decay can take place even at 0°C (1), thus limiting long-term storage. Grey mould is capable of producing ethylene gas (7) which hastens ageing in healthy plant tissues, thereby predisposing them to infection (2).



187 Conidiophores of *Botrytis cinerea* arising from sclerotium

CONTROL Since the fungus can remain viable for long periods, strict hygiene is necessary in both field and store; debris should be removed and destroyed, storage bins disinfected and surfaces cleaned (1,10). The growing crop can be protected by fungicide sprays (3,9). Harvesting of produce intended for long-term storage should be completed before the onset of frosty weather, and care should be taken to minimise mechanical damage during harvesting and handling (2,4,10). Post-harvest fungicides may be applied as a drench (3,4,5) or as a fog (6). Decay can be minimised, though not completely controlled, by rapid cooling and storage at 0°C (1,8). For each batch of harvested plants, a small sample, retained under conditions optimal for decay, permits assessment of the level of infection and hence a fairly accurate prediction of storage potential (9). A refined method, involving microscopic examination of leaf discs, permits a more immediate indication of potential storage life (4). Controlled atmosphere storage inhibits production of ethylene by the fungus, and thereby helps to prolong host resistance (2). Cultivars vary in their susceptibility to grey mould rot (2,4).

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1 BERG L. van den & LENTZ C.P. (1968) The effect of relative humidity and temperature on survival and growth of *Botrytis cinerea* and *Sclerotinia sclerotiorum*. *Canadian Journal of Botany* **46**, 1477–1481.

2 BOCHOW H., HOFMANN K., KUMPF S. & MEINL G. (1976) Über das Anfälligkeitsverhalten verschiedener Kopfkohlsorten während der Lagerung gegenüber *Botrytis cinerea* Pers. *Archiv für Phytopathologie und Pflanzenschutz* **12**, 261–273.

3 BROKENSHIRE T. & PURDIE D.W. (1984) Evaluation of pre-harvest fungicide sprays and post-harvest drenches for the control of *Botrytis cinerea* in stored winter white cabbage. In *Crop Protection in Northern Britain 1984* pp. 406–411.

4 DAVIES R.M. & HEALE J.B. (1985) *Botrytis cinerea* in stored cabbage: the use of germ-tube growth on leaf discs as an indication of potential head rot. *Plant Pathology* **34**, 408–414.

5 GEESON J.D. & BROWNE K.M. (1979) Effect of post-harvest fungicide drenches on stored winter white cabbage. *Plant Pathology* **28**, 161–168.

6 GEESON J.D., ROBINSON R.C. & WILLIAMS D.J. (1982) Evaluation of fungicide fogs for the control of *Botrytis cinerea* in stored winter white cabbage. *Tests of Agrochemicals and Cultivars* No. 3 (*Annals of Applied Biology* **100**, Supplement), 36–37.

7 POAPST P.A., RAMSOOMAIR B.A. & GOURLEY C.O. (1979) On the promotion of senescence in *Brassica oleracea* var. *capitata* by *Alternaria brassicicola* and by *Botrytis cinerea*. *Canadian Journal of Botany* **57**, 2378–2386.

8 POLEGAEV V.I. & PASTUKHOV V.M. (1976) [Effect of temperature and relative humidity of storage environment on infection of cabbages by grey mould rot and bacteriosis.] *Doklady̆ Moskovskoĭ sel'skokhozyaĭstvennoĭ akademii im. K.A. Timiryazeva* No. 221, 131–135.

9 TAHVONEN R. (1983) Short-term, parallel storage in the prediction of storage losses caused by *Botrytis cinerea* Pers. ex Fr. on cabbage. *Journal of the Scientific Agricultural Society of Finland* **55**, 309–314.

10 YODER O.C. & WHALEN M.L. (1975) Factors affecting post-harvest infection of stored cabbage tissue by *Botrytis cinerea*. *Canadian Journal of Botany* **53**, 691–699.

LIGHT LEAF SPOT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Pyrenopeziza brassicae **Sutton & Rawl.**
Conidial state: *Cylindrosporium concentricum* Grev.

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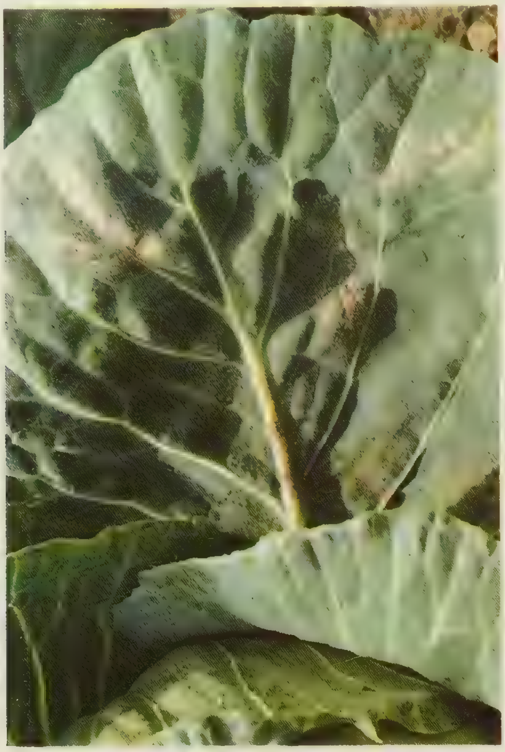

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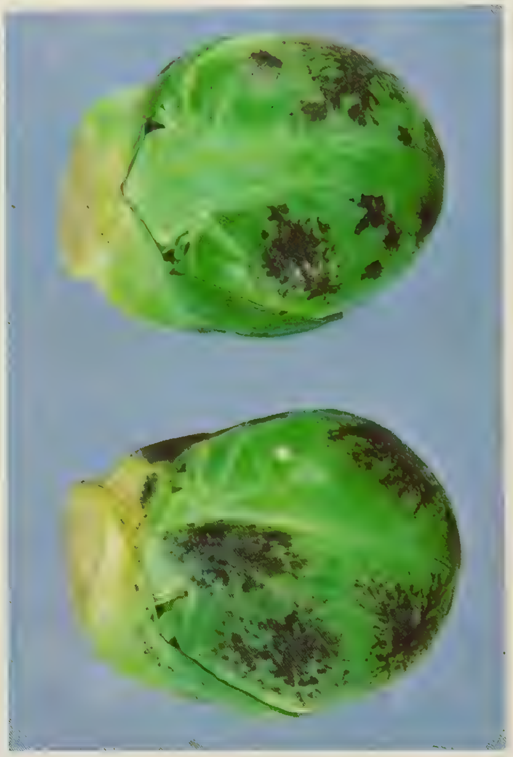
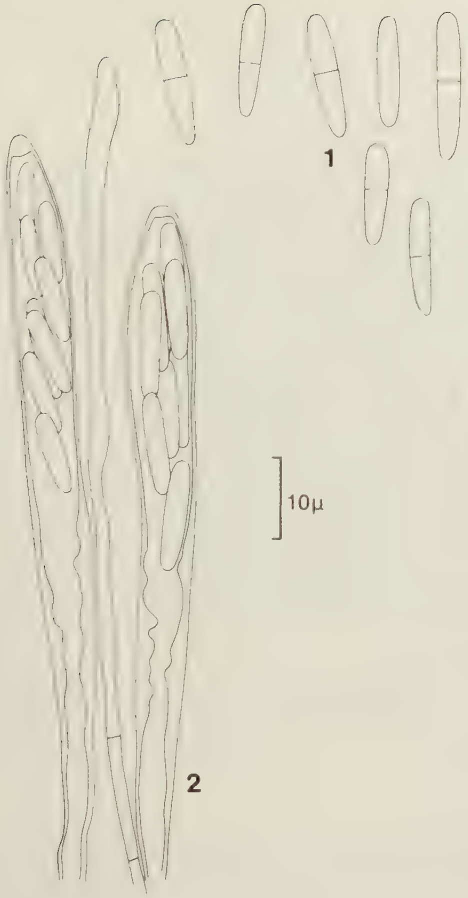


OCCURRENCE This disease has been reported from temperate countries such as the UK (5), Ireland (10), Portugal (1), parts of Australia (3) and New Zealand (2,4). It can cause severe damage to cauliflower (1,2,3) and may occasionally be important on Brussels sprouts (5,10), cabbage (5) and broccoli (2).

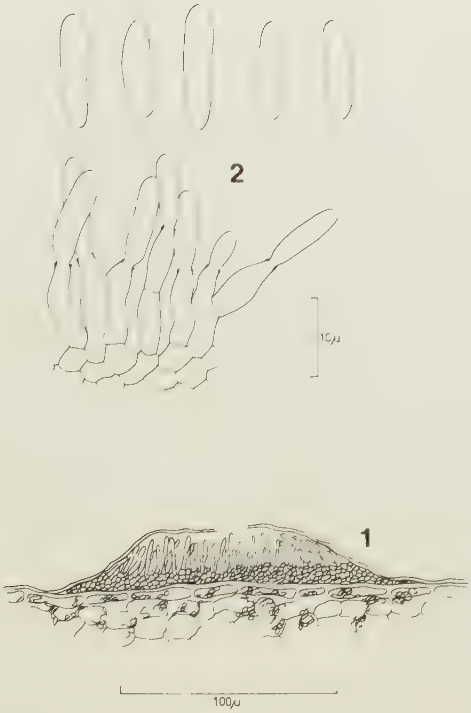
SYMPTOMS Initially lesions consist of bright white spore-masses (of conidia), each usually less than 1 mm in diameter and arranged in concentric circles (3). On the upper leaf surface lesions tend to develop pale blurred centres as spores are displaced. On the lower leaf surface the spore-masses are more likely to remain discrete, darkening with age so that mature lesions appear to consist of black dots surrounded by rings of white dots (5). Infected areas on mid-ribs and leaf stalks are brown and hard and, in cauliflower, the curd may become discoloured (2). In Brussels sprouts the buds are disfigured by darkening lesions.

BIOLOGY The fungus survives in plant debris, on which it may produce the sexual stage (apothecia giving rise to ascospores), these spores being disseminated in air currents (2,6,8,10). Infection occurs during cool wet weather, and fully expanded leaves are more susceptible than young leaves (4,5). The asexual stage (acervuli giving rise to conidia) develops on the growing crop, secondary spore dispersal being chiefly by rain-splash (8). Direct infection occurs (3), especially if the leaf's natural wax coating has been impaired by the use of certain herbicides (8). Epidemics have occurred in recent years (4), and seed crops may suffer infection of pods (5). Although essentially a field disease, light leaf spot can cause losses after harvest because it disfigures the produce (2,5).

CONTROL Rotation with unrelated crops is advisable, and seed treatment has been suggested (7). Weeds do not appear to carry the disease (2,10), but escaped rape plants may be a source of spores (7), oil-seed rape being very susceptible to this disease (6,8). Systemic fungicide sprays should be applied to the growing crop, ordinary fungicides being ineffective (10). After harvest it is essential to ensure prompt destruction of crop debris (7). Some brassica species show resistance to the disease (9).



190 *Pyrenopeziza brassicae* (from *Trans. Br. mycol. Soc.* **71**, 425–439, Rawlinson, Sutton & Muthyalu 1978) 1 ascospores 2 asci and paraphysis



191 *Cylindrosporium concentricum* (from CMI Descr. No. 536, Sutton & Gibson 1977) 1 acervulus 2 conidia and conidiophores

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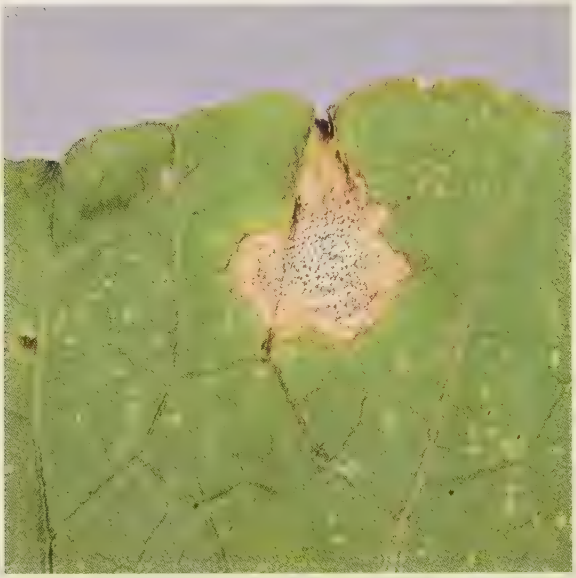
1 CABRAL R.V. de G. (1940) *Gloeosporium concentricum* (Grev.) Berk. et Br. na *Brassica oleracea* L. *Broteria* **9**, 18–41 illus.
2 CHEAH L.-H. & HARTILL W.F.T. (1985) Disease cycle in New Zealand of light leaf spot of brassicas, induced by *Pyrenopeziza brassicae. New Zealand Journal of Agricultural Research* **28**, 567–573 illus.
3 HALSEY F.J. (1934) A disease of cauliflowers in Victoria, Australia (*Gloeosporium concentricum* (Grev.) Berk. and Br.). *Proceedings of the Royal Society of Victoria NS* **47**, 96–111 illus.
4 HARTILL W.F.T. & CHEAH L.-H. (1984) Some effects of climate and plant growth on the development of light leaf spot in cauliflowers. *New Zealand Journal of Agricultural Research* **27**, 441–449.
5 HICKMAN C.J., SCHOFIELD E.R. & TAYLOR R.E. (1955) Light leaf spot of brassicas. *Plant Pathology* **4**, 129–131 illus.
6 LACEY M.E., RAWLINSON C.J. & McCARTNEY H.A. (1987) First record of the natural occurrence in England of the teleomorph of *Pyrenopeziza brassicae* on oilseed rape. *Transactions of the British Mycological Society* **89**, 135–140 illus.
7 MADDOCK S.E. & INGRAM D.S. (1981) Studies of survival and longevity of the light leaf spot pathogen of brassicas, *Pyrenopeziza brassicae. Transactions of the British Mycological Society* **77**, 153–159.
8 RAWLINSON C.J., SUTTON B.C. & MUTHYALU G. (1978) Taxonomy and biology of *Pyrenopeziza brassicae* sp. nov. (*Cylindrosporium concentricum*), a pathogen of winter oilseed rape (*Brassica napus* ssp. *oleifera*). *Transactions of the British Mycological Society* **71**, 425–439 illus.
9 SIMONS A.J. & SKIDMORE D.I. (1988) Race-specific resistance to light leaf spot in *Brassica oleracea. Transactions of the British Mycological Society* **90**, 431–435.
10 STAUNTON W.P. (1967) Studies on light leaf spot (*Gloeosporium concentricum*) of Brassicae. *Irish Journal of Agricultural Research* **6**, 203–211 illus.

PHOMA ROT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Leptosphaeria maculans **(Desm.) Ces. & de Not.**

Conidial state: *Phoma lingam* (Tode:Fr.) Desm.

192



OCCURRENCE This disease is widespread in temperate countries such as the UK (4), Sweden (6), Germany and the Netherlands (1). It has been recorded in the USA (2,3), Canada (5) and Australia, and may also occur in tropical countries possessing a cool wet season (CMI Map 73).

SYMPTOMS Pale brown circular spots develop on the leaves; mature lesions may be more than 1 cm across and are characterised by the presence of numerous minute black bodies (pycnidia) clearly visible to the naked eye. In stored cabbage the butt may be disfigured by a ring of black sunken spots (infected vascular strands); subsequently a dry basal rot develops, with pycnidia abundant in diseased tissue (page 341) (1).

BIOLOGY The fungus is seed-borne (2,4,5) and under moist conditions there is rapid spread of the disease as spores (conidia) are splash-dispersed in the seed-bed. Seedlings infected shortly before transplanting escape detection and transmit the fungus to the field. In the presence of moisture (from rain, irrigation or dew) direct penetration of leaf tissue occurs, with resultant spotting and production of more spores. If leaf stalks are infected, the fungus usually grows down and into the main stem, hence the common name of 'blackleg'. Further spread is likely at harvest time when knives become contaminated. Post-harvest rotting can be severe in non-refrigerated stores (1). Although the fungus commonly exists in the asexual state (pycnidia giving rise to conidia), it may also produce the sexual state (pseudothecia giving rise to ascospores). Wind-dispersed ascospores may be responsible for field to field infection. The fungus can survive in crop debris in the soil, and on contaminated equipment and footwear (1).

CONTROL Since the fungus may remain viable for several years, a long crop rotation is desirable, and it should be borne in mind that other cruciferous crops, such as swedes, turnips and rape, are also susceptible to this disease (4). It is essential to sow clean or treated seed (1,4) and to ensure that the seed-bed site is free of contamination. Fungicide sprays may be applied to the growing crop (1,3). If the weather is wet around harvest time there is a risk of widespread infection unless the crop is promptly gathered into store (1). Strict hygiene is necessary to limit the spread of disease via contaminated soil and implements; crop residues should be promptly burned (3). Work is in progress to breed resistant cultivars (6).

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1 BAKEL J.M.M. van (1968) Vallers en kanker in bewaarkool. *Mededelingen Proefstation voor de Groenteteelt in de Vollegrond in Nederland* **41**, 31 pp illus.

2 BONMAN J.M., GABRIELSON R.L., WILLIAMS P.H. & DELWICHE P.A. (1981) Virulence of *Phoma lingam* to cabbage. *Plant Disease* **65**, 865–867 illus.

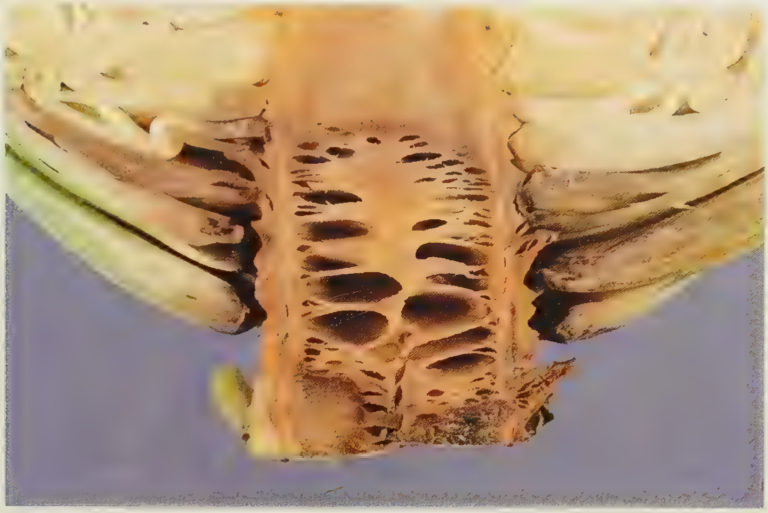
3 GREATHEAD A.S. (1984) Control of blackleg disease of broccoli through cultural practices and the application of fungicides. *Phytopathology* **74**, 828 [Abstract].

4 MAUDE R.B., HUMPHERSON-JONES F.M. & SHURING C.G. (1984) Treatments to control *Phoma* and *Alternaria* infections of brassica seeds. *Plant Pathology* **33**, 525–535.

5 PETRIE G.A. (1988) The rapid differentiation of virulent and weakly virulent strains of *Leptosphaeria maculans* (blackleg or stem canker) and related pycnidial fungi from *Brassica* seeds and stems. *Canadian Journal of Plant Pathology* **10**, 188–190.

6 SJÖDIN C. & GLIMELIUS K. (1988) Screening for resistance to blackleg *Phoma lingam* (Tode ex Fr.) Desm. within Brassicaceae. *Journal of Phytopathology* **123**, 322–332.

PHYTOPHTHORA ROT of cabbages and Chinese cabbages caused by ***Phytophthora porri*** **Foister**



193

OCCURRENCE This disease has been recorded on cabbages in Norway (5), the Netherlands and the UK (1,3), and on Chinese cabbages in West Germany (4).

SYMPTOMS Grey watersoaked blotches may appear on the wrapper leaves. More usually, however, decay begins at the stalk or butt and progresses into the head (1,5). Rotted tissue is brown to dark grey, with a firm texture and a distinctive sour odour. Cavities tend to form in the stalk tissue, and sparse white mould strands may be observed, though spores have not been recorded on infected tissue of harvested vegetables. Bacterial soft rot (q.v.) frequently follows (1,5).

BIOLOGY The fungus survives in the soil or on crop debris as thick-walled resting spores (chlamydospores) (2). It can also persist in the sexual state (oospores) (5). Under wet conditions the asexual stage is produced (sporangia which may give rise to zoospores), and dissemination is by water-splash. Infection probably occurs during harvest, since rain at this time is associated with serious losses in store (5). It is likely that the fungus can be transmitted on knives, since disease incidence has been observed to increase after mid-storage trimming (1). The optimal growth temperature for the fungus lies between 15° and 20°C, but during prolonged storage it can make significant progress even at 0°C (5). Eventually entire heads are invaded, but the decay only rarely spreads into adjacent sound heads.

CONTROL Since fungicides are largely ineffective against this disease, control is by cultural practices, the most important of which is prompt destruction of any infected plant debris. Crop rotation is desirable, and it should be noted that leeks and onions are also susceptible to infection by this fungus (2). If there is rain around harvest time, it is advisable to arrange for hygienic trimming to take place under shelter, before the commencement of storage (5). Refrigeration serves to minimise the rate of disease development in store.

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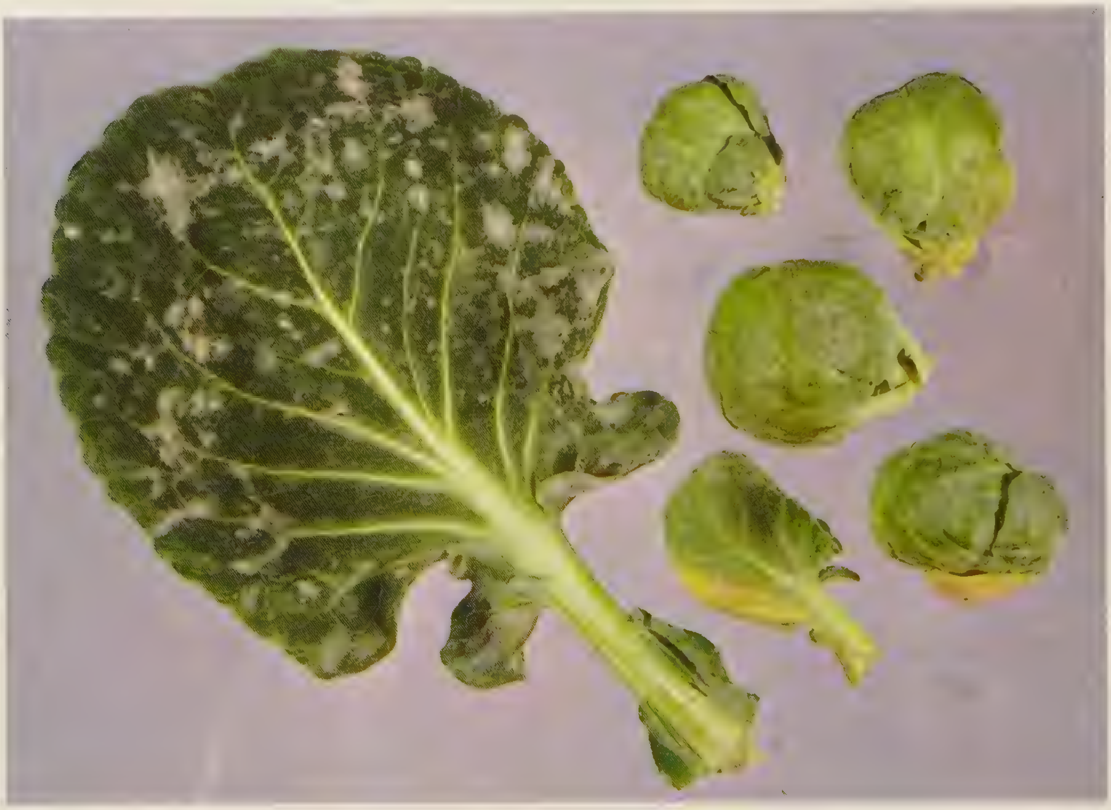
1 GEESON J.D. (1976) Storage rot of white cabbage caused by *Phytophthora porri*. *Plant Pathology* **25**, 115–116 illus.
2 KATSURA K. & MAEDA S. (1971) [Chlamydospore formation of *Phytophthora porri* Foister.] *Scientific Reports of Kyoto Prefectural University (Agriculture)* No. 23, 34–38 illus.
3 KEAR R.W., WILLIAMS D.J. & STEVENS C.C. (1977) The effect of iprodione on the fungal deterioration of stored white cabbage. *Proceedings of the 1977 British Crop Protection Conference, Brighton,* 189–195.
4 KIEWNICK L. (1985) *Phytophthora porri* Foister als Ursache einer Lagerfäule bei Chinakohl. *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* **37**, 136–137 illus.
5 SEMB L. (1971) A rot of stored cabbage caused by a *Phytophthora* sp. *Acta Horticulturae* No. 20, 32–35 illus.

POWDERY MILDEW of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Erysiphe cruciferarum **Opiz ex Junell**

Conidial state: *Oidium* sp.

194

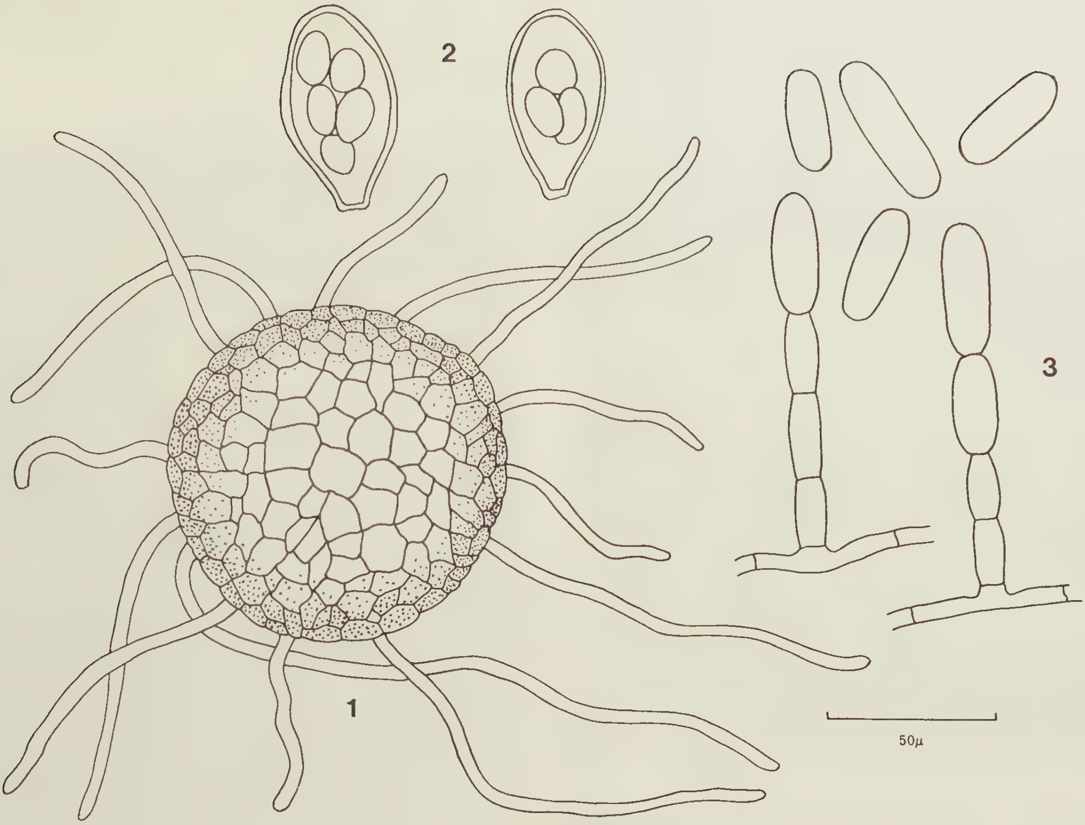


OCCURRENCE Powdery mildew is common in semi-arid regions such as the Middle East (1,5) and parts of India (7,8), but also causes occasional losses in temperate countries after a warm dry spell (10). It has been recorded in the USA (10), Japan (9), Sweden (4) and Yugoslavia (2) and, although generally of minor importance, has resulted in serious losses in Brussels sprouts grown in the UK (3).

SYMPTOMS In partially resistant cultivars the sole symptom may be minute black flecks on the leaves (10). In susceptible cultivars small patches of whitish mould develop on the leaves, usually appearing first on the upper surface. Powdery greyish-white spores (conidia) develop, and eventually the entire leaf may be completely covered. In Brussels sprouts the fungus may also penetrate deeply into the buds and, at low temperatures, lesions on the buds tend to turn black (3).

BIOLOGY The fungus only rarely produces the sexual stage (cleistothecia giving rise to ascospores) (4,7,8), and usually survives as mould strands, which require living vegetative tissue if they are to remain viable. This condition is frequently met, however, since cruciferous crops are commonly in the ground for up to a year or, if grown for seed, two years (3). Asexual spores (conidia) are dispersed by wind and, in contrast to those of most other fungi, are capable of causing infection even in a dry atmosphere (10), by direct penetration of intact tissue. A strain which infects cabbage does not necessarily infect cauliflower (9), and the extent of spread between related crops is not known (3). Although primarily a field disease, powdery mildew may result in post-harvest losses, for example entire consignments of Brussels sprouts may be rejected as unfit for processing (3).

CONTROL Preventive fungicide sprays may be advisable at certain times of year. There have been attempts at biological control, using a species of harmless fungus which inhibits powdery mildew development (2). There has been some success in breeding resistant cultivars of cabbage (10) and Brussels sprouts (3,6).



195 *Erysiphe cruciferarum* (from CMI Descr. No. 251, Purnell & Sivanesan 1970)
1 cleistothecium 2 asci and ascospores 3 conidia and conidiophores

1 AL-HASSAN K.K. (1973) Powdery mildews and their hosts. *FAO Plant Protection Bulletin* **21**, 88–91.

2 CVJETKOVIĆ B. & MANDIĆ R. (1980) [*Erysiphe cruciferarum,* the pathogen of powdery mildew of cabbage and its hyperparasite *Ampelomyces quisqualis* Ces.] *Zaštita Bilja* **31**, 373–377.

3 DIXON G.R. (1974) Field studies of powdery mildew (*Erysiphe cruciferarum*) on Brussels sprouts. *Plant Pathology* **23**, 105–109 illus.

4 JUNELL L. (1967) A revision of *Erysiphe communis* (Wallr.) Fr. sensu Blumer. *Svensk Botanisk Tidskrift* **61**, 209–230 illus.

5 ORAN Y.K. (1974) Host range and distribution of the powdery mildews in Turkey. *Journal of Turkish Phytopathology* **3**, 1–27.

6 RUDGARD S.A. & WHEELER B.E.J. (1985) The development of *Erysiphe cruciferarum* on field-grown Brussels sprouts and associated changes in soluble amino-acids in foliage leaves. *Plant Pathology* **34**, 616–625.

7 SAHARAN G.S. & KAUSHIK J.C. (1981) Occurrence and epidemiology of powdery mildew of *Brassica. Indian Phytopathology* **34**, 54–57.

8 SANKHLA H.C., SINGH H.G., DALELA G.G. & MATHUR R.L. (1967) Occurrence of perithecial stage of *Erysiphe polygoni* on *Brassica campestris* var. *sarson* and *B. juncea. Plant Disease Reporter* **51**, 800.

9 SAWAMURA K. (1957) [On the powdery mildew of cabbage.] *Annals of the Phytopathological Society of Japan* **22**, 166–167 illus.

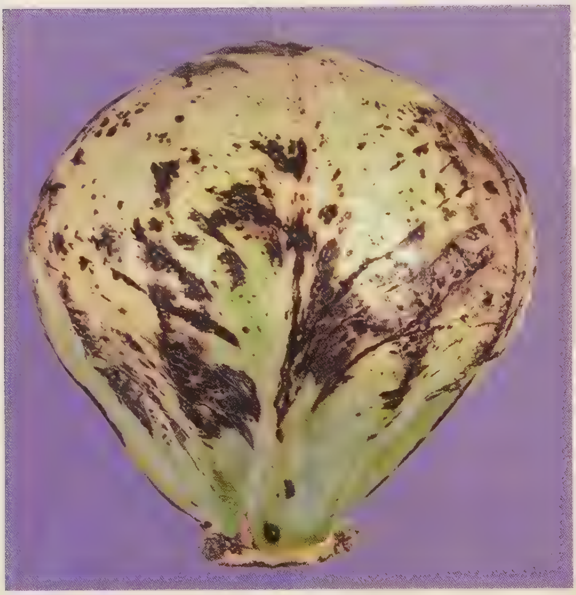
10 WALKER J.C. & WILLIAMS P.H. (1965) The inheritance of powdery mildew resistance in cabbage. *Plant Disease Reporter* **49**, 198–201.

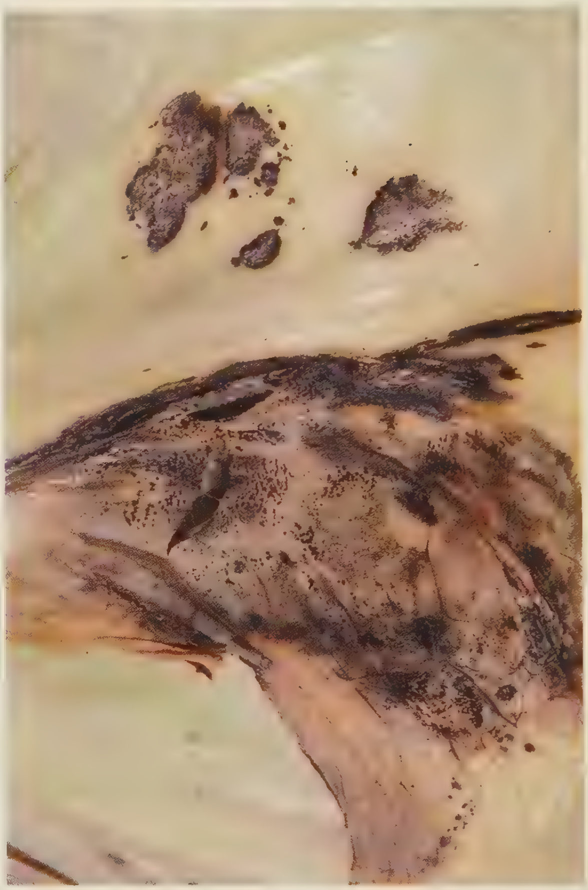
RING SPOT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Mycosphaerella brassicicola **(Duby) Lindau**

Spermatial state: *Asteromella brassicae* (Chev.) Boerema & v. Kest.

196





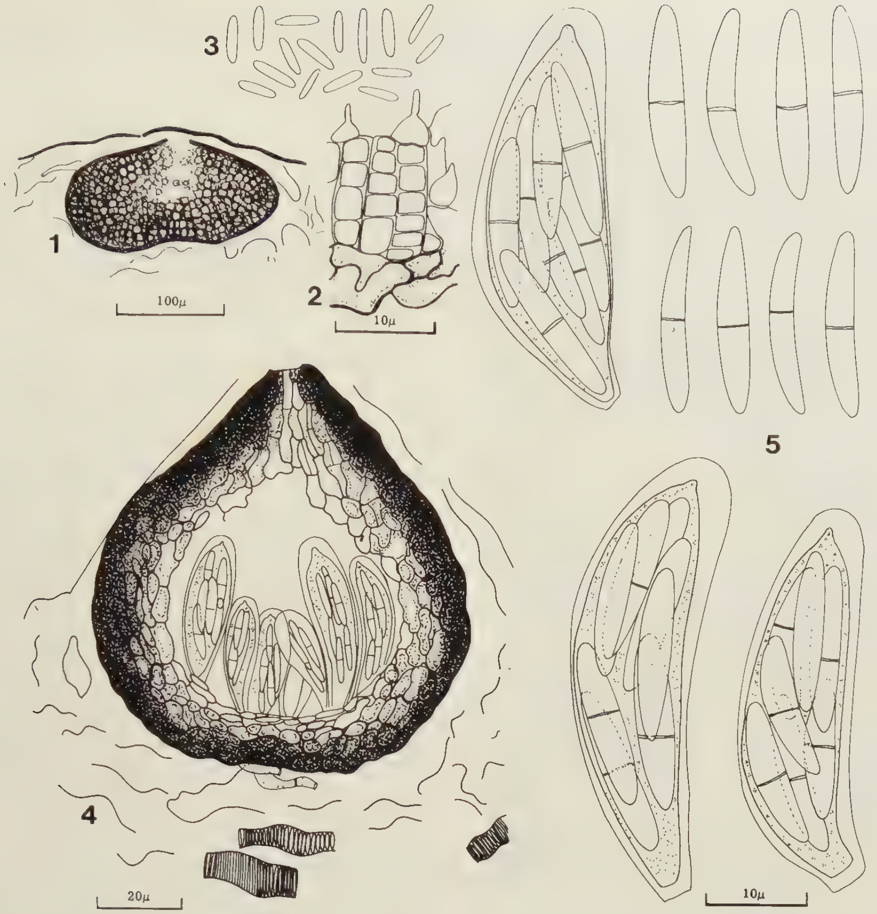
197 Close-up showing pycnidia (spermagonia)

OCCURRENCE Ring spot is widespread (CMI Map 189) but is chiefly important in cool temperate countries such as the UK (2), France (4), Germany (7), the Netherlands (6), Denmark, New Zealand (3), parts of Australia (10), and coastal regions of the USA (5,9).

SYMPTOMS In addition to circular spots up to about 1 cm in diameter, small irregular lesions may develop (7,9). The former may show concentric zones of brown and grey with a well-defined border, hence the common name of 'ring spot' (5,9). Frequently, however, affected areas are irregular in shape and almost uniformly dark, with a dry leathery texture. Minute dark brown bodies (pseudothecia or spermagonia or both) develop within the affected area (2,5,8,9)

BIOLOGY The fungus survives in crop debris (7) in the sexual state (pseudothecia giving rise to ascospores) and the spores are disseminated in air currents (6,8,9). In a cool moist environment mature leaves (3) are infected via natural openings (stomata) (9), optimal conditions for disease development being a temperature of 16° to 20°C coupled with a saturated atmosphere (5). Infection may also occur via wounds sustained at harvest time. The other phase in the life cycle of this fungus (spermagonia giving rise to spermatia) plays no part in infection (1,2,8). Symptoms continue to develop in store; at 0°C enlargement of spots is insignificant during a brief transit period (9) but substantial deterioration can occur during long-term storage. In the seed crop, pods may be invaded (4,9), though the importance of seed-borne infection is unclear.

CONTROL Crop rotation is desirable; it should be noted that the disease has also been reported on turnip (9) and rape. Seed treatment has been recommended, followed by strict hygiene in seed-bed and field (7). Fungicide spraying (4,10) should be started as soon as the first-formed leaves are fully expanded (3). Post-harvest fungicide treatment should be followed by prompt cooling and low temperature storage.



198 *Mycosphaerella brassicicola* (from CMI Descr. No. 468, Punithalingam & Holliday 1975) 1 pycnidium (spermagonium) 2 part of pycnidial (spermagonial) wall and conidia (spermatial cells) 3 conidia (spermatial cells) 4 pseudothecium 5 asci and ascospores

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1 BOEREMA G.H. & KESTEREN H.A. van (1964) The nomenclature of two fungi parasitising *Brassica. Persoonia* **3**, 17–28 illus.

2 DRING D.M. (1961) Studies on *Mycosphaerella brassicicola* (Duby) Oudem. *Transactions of the British Mycological Society* **44**, 253–264 illus.

3 HARTILL W.F.T. & SUTTON P.G. (1980) Inhibition of germination of *Mycosphaerella brassicicola* ascospores on young cabbage and cauliflower leaves. *Annals of Applied Biology* **96**, 153–161.

4 JOUAN B., LEMAIRE J.-M. & HERVÉ Y. (1972) Étude des maladies du chou-fleur (*Brassica oleracea* L. var. *botrytis* DC). 1. Les parasites des porte-graines de chou-fleur d'hiver. *Annales de Phytopathologie* **4**, 133–155 illus.

5 NELSON M.R. & POUND G.S. (1959) The relation of environment to the ringspot (*Mycosphaerella brassicicola*) disease of crucifers. *Phytopathology* **49**, 633–640 illus.

6 QUAK F. (1957) Bladvlekkenziekte bij spruitkool veroorzaakt door *Mycosphaerella brassicicola* (Fr.) Lindau. *Mededelingen Directeur van de Tuinbouw* **20**, 317–320.

7 RUDNICK M. (1986) Epidemisches Auftreten der Ringfleckenkrankheit an Kohl in Schleswig-Holstein, verursacht durch *Mycosphaerella brassicicola* (Duby) Lindau. *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* **38**, 83–85 illus.

8 SNYDER W.C. (1946) Spermogonia versus pycnidia in *Mycosphaerella brassicicola. Phytopathology* **36**, 481–484 illus.

9 WEIMER J.L. (1926) Ringspot of crucifers caused by *Mycosphaerella brassicicola* (Fr.) Lindau. *Journal of Agricultural Research* **32**, 97–132 illus.

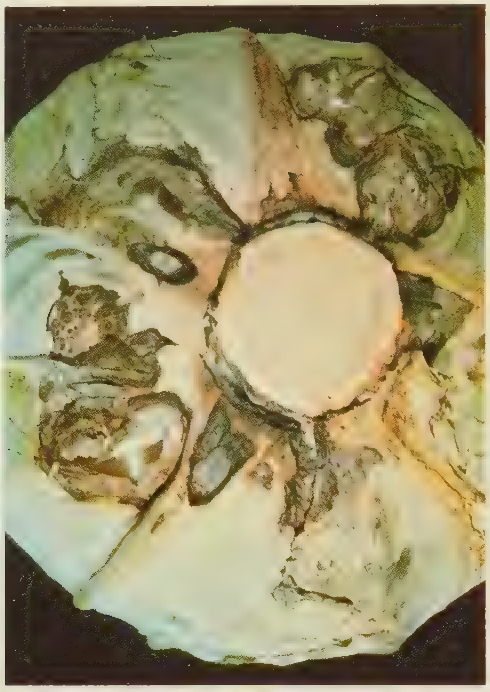
10 WICKS T., LOMMAN G. & ROGERS I.S. (1987) Fungicide control of ringspot (*Mycosphaerella brassicicola*) in Brussels sprouts. *Australian Journal of Experimental Agriculture* **27**, 597–600.

RHIZOCTONIA ROT of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Thanatephorus cucumeris **(Frank) Donk**

Sclerotial state: *Rhizoctonia solani* Kühn

199



OCCURRENCE This disease has been reported on cabbages in Germany, various parts of the USA (1,4,5), and recently on cabbages in India and Pakistan. It has also been observed on cauliflowers in Italy (2), and on Chinese cabbages in the USA, South Korea and the West Indies (3).

SYMPTOMS In the so-called 'bottom rot' elongated sunken black lesions develop on the undersurface of the midribs, while circular spots appear on the lower part of the leaves (4). Alternatively, or in addition, small irregularly shaped lesions may develop on the tops of the heads, gradually coalescing and darkening (1,4). Eventually a 'head rot' may ensue. As decay progresses, a dark web-like mould (3) spreads over the surface, giving rise to a mass of flat brown resting bodies (sclerotia) up to 0.5 cm across.

BIOLOGY The fungus persists as sclerotia in the soil or in infected crop debris. After a period of warm wet weather it may produce the sexual stage (basidia giving rise to basidiospores), the spores being formed on a brownish-grey mat-like mould which grows over the soil and up the stem. These spores are dispersed by wind, but their significance in the infection process is unclear (1). The other stage (sterile mould giving rise to sclerotia) is also favoured by moist conditions, and the mould can attack plants at all stages of their development. The optimal temperature for disease development lies between 25° and 30°C (1,4). The fungus grows scarcely at all below 10°C (4), but infected cold-stored cabbage is reported to be especially prone to secondary bacterial soft rot (q.v.) of the outer leaves (1).

CONTROL Since the disease is soil-borne, control measures include sterilisation of the seed-bed, followed by careful husbandry in the field so as to minimise contact between the lower leaves and the ground. (It was found that if, during cultivations, soil was banked up around the plant stalk and base of the head, the resulting high humidity facilitated direct infection of the lower leaves.) After harvest, infected plant debris should be promptly destroyed. Crop rotation can be advantageous, since strains of the fungus attacking other crops do not necessarily attack crucifers (1,5). Some cabbage cultivars are resistant to the disease (5).

1 ABAWI G.S. & MARTIN S.B. (1985) Rhizoctonia foliar blight of cabbage in New York State. *Plant Disease* **69**, 158–161 illus.

2 D'ERCOLE N. & NIPOTI P. (1978) La rizottoniosi del cavolfiore. 3. Modalità di penetrazione dell'agente patogeno (*Rhizoctonia solani* Kühn). *Informatore Fitopatologico* **28**(9), 21–23 illus.

3 RAJNAUTH G. (1987) Web blight, an important disease of bean and pak-choi in Trinidad. *Tropical Agriculture* **64**, 356–358.

4 WELLMAN F.L. (1932) *Rhizoctonia* bottom rot and head rot of cabbage. *Journal of Agricultural Research* **45**, 461–469 illus.

5 WILLIAMS P.H. & WALKER J.C. (1966) Inheritance of *Rhizoctonia* bottom rot resistance in cabbage. *Phytopathology* **56**, 367–368 illus.

See also:

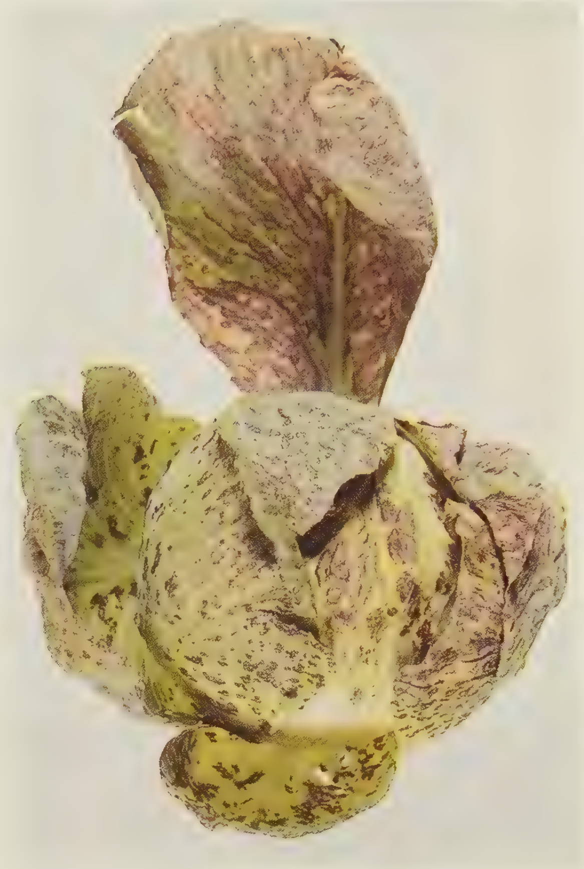
SEIDEL D. & DAEBELER F. (1963) *Rhizoctonia solani* als Erreger einer Kopffäule am Weisskohl. *Nachrichtenblatt für den Deutschen Pflanzenschutzdienst* **17**, 43 illus.

VIRUS DISEASES of cabbages, kales, cauliflowers and Chinese cabbages

OCCURRENCE The most important are those caused by turnip mosaic virus (TuMV) and cauliflower mosaic virus (CaMV), both of which have worldwide distribution. Records include China, Japan and the Philippines (3), the USA (1), the UK (2,4) and West Germany (5). The two viruses frequently occur together, and many different strains exist (1,3).

SYMPTOMS TuMV induces dark zoned spots on the outer leaves of cabbage and kale, hence the earlier name of 'cabbage black ringspot virus'. Black lesions 5–10 mm across (cf. 'pepper spotting', q.v.) also develop internally in heads of stored cabbage (5). Another symptom is mottling or 'mosaic', characterised by pale blotches (1). CaMV may induce any of the above symptoms; alternatively it may cause 'vein-clearing', in which the veins of the outer leaves become pale (1). In Chinese cabbage infected with TuMV the heart leaves are characteristically crinkled and stunted. Virus infections of cauliflower, Brussels sprouts and broccoli are largely confined to outer leaves, which are normally removed at harvest.

BIOLOGY The viruses persist throughout the year in brassica crops and possibly also in ornamentals and weeds belonging to the cabbage family (3). Transmission is by aphids. These insects pierce the surface of leaves and stems with their mouth parts, in order to suck out the sap; in doing so they acquire virus particles and subsequently introduce them into healthy plants. Disease incidence is therefore determined by insect numbers and movement. These are much influenced by seasonal factors; mild winters favour substantial early migrations of aphids and hence severe disease outbreaks. Lesions on the outer leaves of cabbage at harvest time are usually indicative of the incidence of internal lesions developing during storage.



200 Turnip mosaic virus of cabbage

CONTROL Seed-beds should be sited away from existing brassica crops. It is difficult to achieve control solely by the use of pesticides, because virus infection may have taken place in the early phase of infestation before spraying has begun (2). Present recommendations include the use of resistant cultivars (2,3).

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1 KHAN M.A. & DEMSKI J.W. (1982) Identification of turnip mosaic and cauliflower mosaic viruses naturally infecting collards. *Plant Disease* **66**, 253–256 illus.

2 KNIGHT C., WRIGHT I.R. & PRIESTLEY R.H. (1984) Resistance of winter white cabbage varieties to turnip mosaic virus. *Journal of the National Institute of Agricultural Botany* **16**, 555–559 illus.

3 PROVVIDENTI R. (1980) Evaluation of Chinese cabbage cultivars from Japan and the People's Republic of China for resistance to turnip mosaic virus and cauliflower mosaic virus. *Journal of the American Society for Horticultural Science* **105**, 571–573.

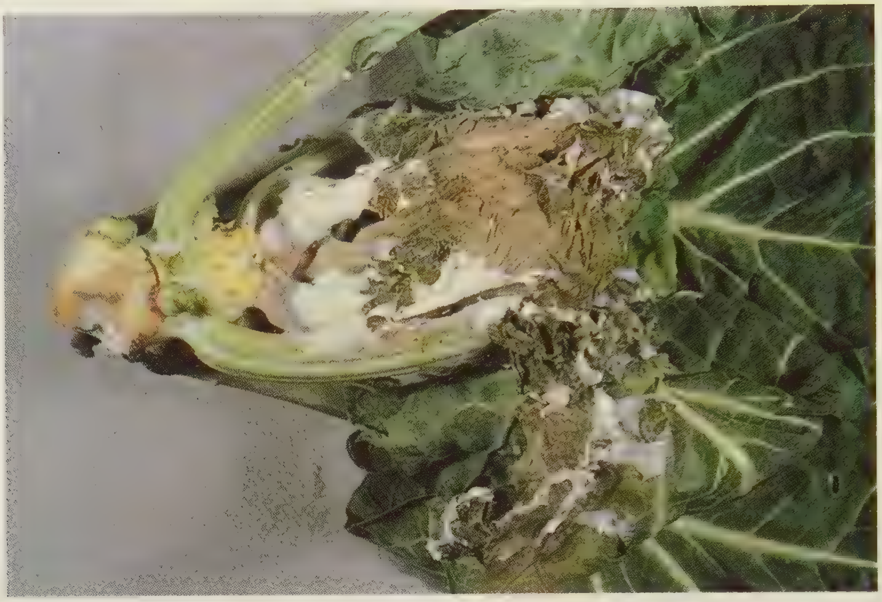
4 PINK D.A.C. & WALKEY D.G.A. (1988) The reaction of summer-maturing and autumn-maturing cauliflowers to infection by cauliflower mosaic and turnip mosaic viruses. *Journal of Horticultural Science* **63**, 95–102.

5 WEIDEMANN W.-L. (1981) Virusbedingte Innenblattnekrosen bei Weisskohl. *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* **33**, 17–20 illus.

WATERY SOFT ROT of cabbages, kales, cauliflowers, Brussels sprouts, broccoli and Chinese cabbages caused by

Sclerotinia minor **Jagger**
Sclerotinia sclerotiorum **(Lib.) de Bary**

201

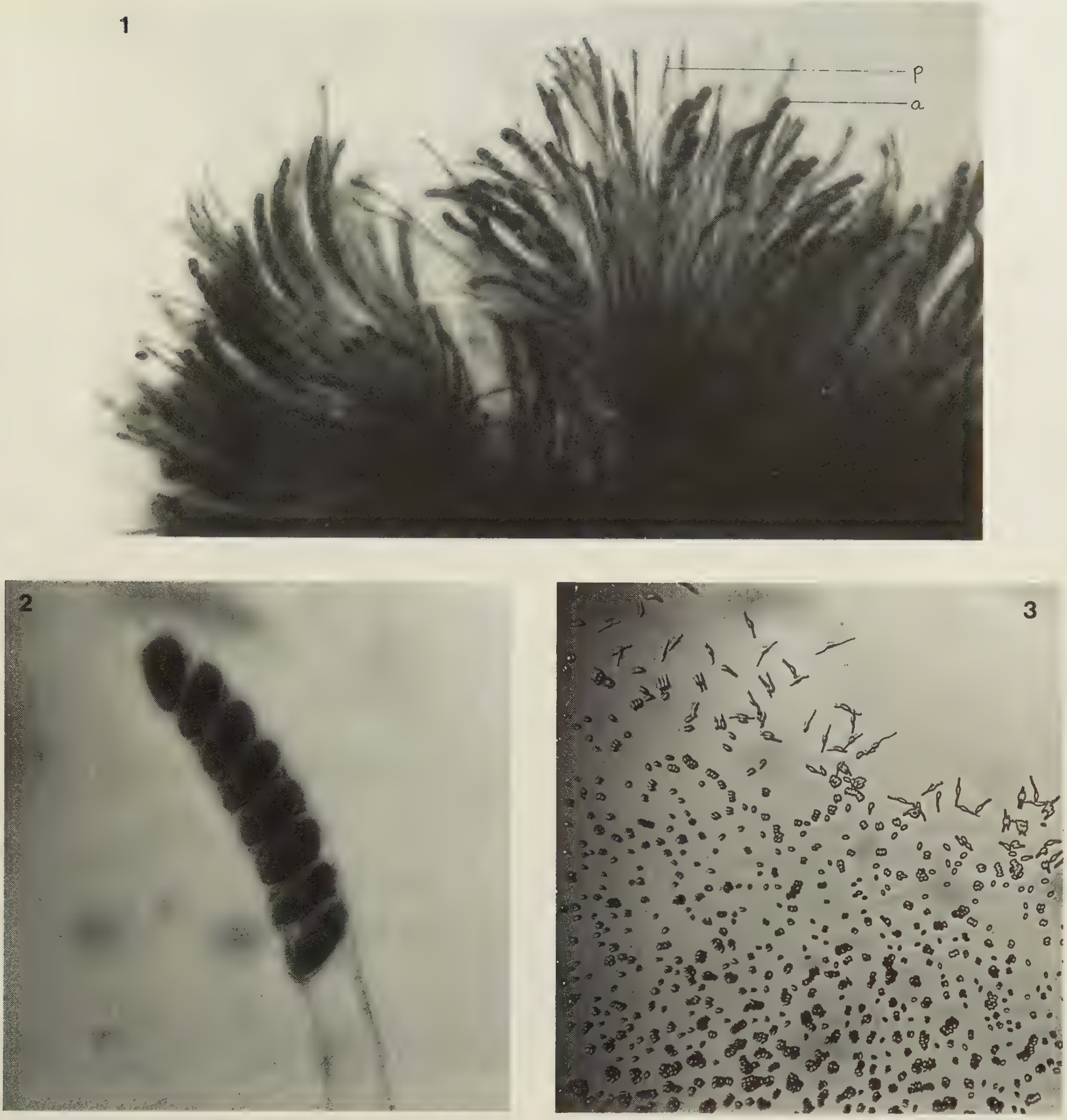


OCCURRENCE Records of this disease include cabbage in Italy (4), cauliflower in Australia (3) and India (5), and cabbage, Brussels sprouts, broccoli and Chinese cabbage in the USA (1,2). In Australia (New South Wales) *Sclerotinia minor* tends to prevail in inland areas during summer when a dry period is followed by rain or irrigation; *S. sclerotiorum*, in contrast, is more prevalent in coastal regions under cool, moist conditions in autumn, winter or spring (3).

SYMPTOMS Lesions are at first pinkish-brown and watersoaked; in contrast to bacterial soft rot (q.v.) there is no disagreeable odour. Under dry conditions the rot may remain firm, but more usually there is a soft breakdown with much leakage of liquid. Small black bodies (sclerotia) develop on and within the tissue, those of *S. minor* being 0.5 to 2 mm and those of *S. sclerotiorum* 5 to 10 mm.

BIOLOGY These fungi survive on weeds or as resting bodies (sclerotia) in soil and crop debris. *S. minor* is capable of causing infection within a few days of the start of rainfall or irrigation, by mould strands growing through and over the soil; they attack the stalk of the plant, or leaves which are in contact with the soil (4). If moist conditions persist for several weeks, *S. sclerotiorum* produces the sexual stage (apothecia giving rise to ascospores) (3). The spores are forcibly ejected and can cause infection of leaves or stems, provided that moribund tissue or a wound is present (1). Flowering weeds (themselves infected by way of dying blossoms) can act as a source of infection of healthy plants in contact (2). Plants can undergo a sudden collapse in the field, the so-called 'drop' (cf. lettuce, q.v.) (1), or they may be harvested in apparently sound condition and rot in store. Each of the fungi is capable of inducing a watery breakdown of tissue, the optimal temperature for decay being approximately 20°C. Fungal growth is significant even at 0°C, and is therefore a limiting factor in long-term storage. Crops grown for seed are particularly susceptible to attack by *S. sclerotiorum* because their senescent flowers provide a means of entry for the fungus. Seed may be infected internally or else be contaminated with sclerotia.

CONTROL It is important to sow clean seed. Weeds should be controlled, both because they may harbour the fungi and because their foliage creates a moist environment favouring disease development. Soil disinfection may be feasible, and crops can be sprayed with fungicide (4), although it is sometimes difficult to obtain effective coverage of the undersides of plants (3). Infected plant debris should be promptly removed and destroyed. In India recommended cultural measures include removing infected leaves at weekly intervals (5) and mulching with pine needles or sunflower head residues.



202 *Sclerotinia sclerotiorum* 1 asci (a) and paraphyses (p) 2 ascus and ascospores 3 germinating ascospores

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1 DAVIS W.H. (1925) Drop of Chinese cabbage and our common cabbage caused by *Sclerotinia sclerotiorum* (Lib.) Massee (*Sclerotinia libertiana* Fckl.). *Phytopathology* **15**, 249–259 illus.

2 DILLARD H.R. & HUNTER J.E. (1986) Association of common ragweed with sclerotinia rot of cabbage in New York State. *Plant Disease* **70**, 26–28 illus.

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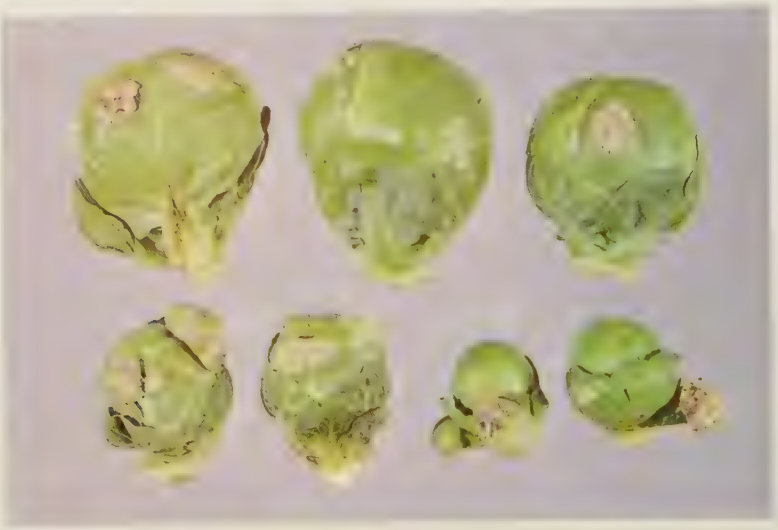
WHITE BLISTER of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages caused by

Albugo candida **(Pers. ex Hooker) O. Kuntze**

203



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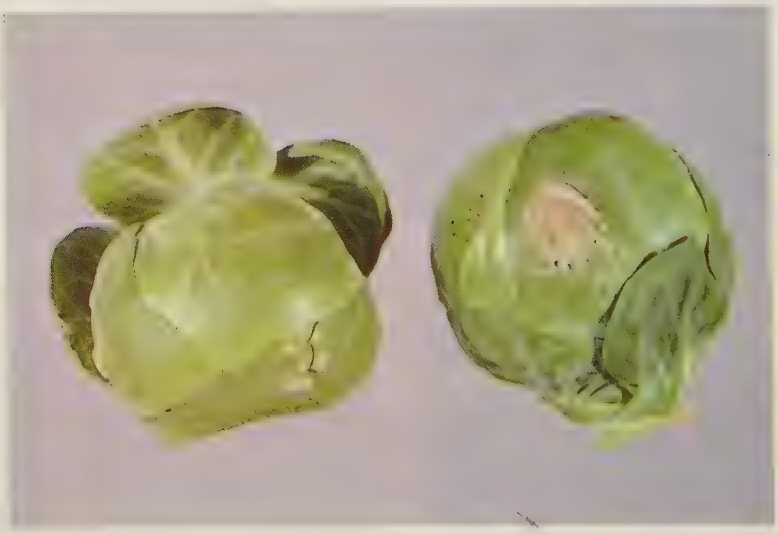
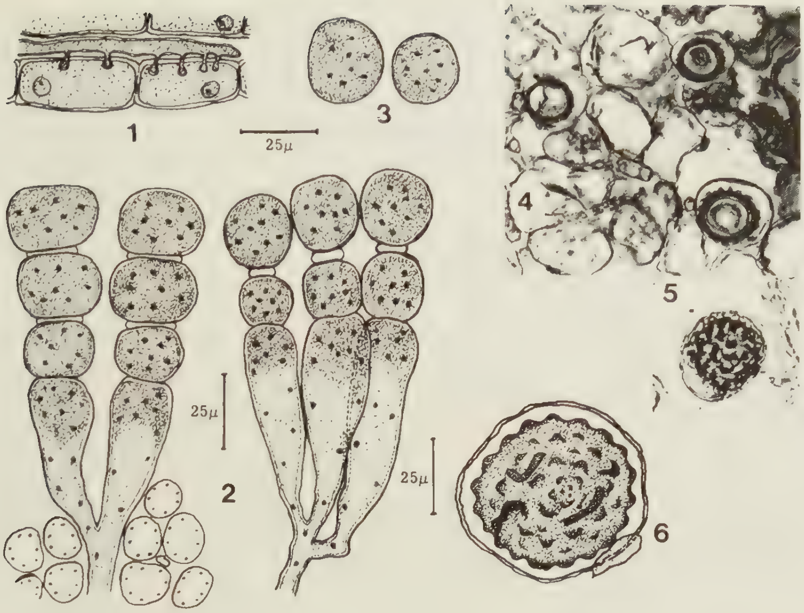


OCCURRENCE White blister is widespread but generally of minor importance. It has been recorded on cabbage, kale, broccoli and Chinese cabbage in the USA (2), on cabbage, kale, broccoli and Brussels sprouts in the UK (1,4), on cauliflower in France and Romania (7), on kale in Kenya, and on Chinese cabbage in India (8), the Philippines (5) and Japan (10). The disease also occurs in Italy and Canada (3,6,9).

SYMPTOMS Raised white (or, rarely, pale yellow) pustules develop on the leaves, eventually rupturing and becoming powdery (owing to the formation of sporangia) (3). White blister is frequently associated with downy mildew (q.v.) (5,7).

BIOLOGY The fungus survives in the sexual state (oospores) (9) or as mould strands in living host tissue; either can give rise to the asexual stage (sporangia). The sporangia are dispersed by air currents and, if they remain dry for a short period, give rise to asexual spores (zoospores). Subsequent wet conditions permit spore germination and infection of leaves via their natural openings (stomata) (4). The optimal temperature for disease development lies between 15° and 20°C. Harvested plants are reduced in value by the disfiguring lesions; furthermore, in the seed crop the fungus may spread to the flower head and contaminate the seed (7).



205 *Albugo candida* (from CMI Descr. No. 460, Mukerji 1975) 1 haustoria 2 sporangiophores with sporangial chains 3 sporangia 4 oospores in infected tissue × 120 5 mature oospore with persistent oogonial wall × 200 6 mature oospore

CONTROL Clean or treated seed should be used (9), and care should be taken to ensure correct use of fertiliser (7). Soil treatment has been recommended, and fungicide sprays may be used on the developing crop (7,9). Several races of the fungus exist, and those which attack weeds are generally not capable of attacking crops (2,4). Some resistant cultivars are available (1,6,8).

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5 OCFEMIA G.O. (1925) The occurrence of the white rust of crucifers and its associated downy mildew in the Philippines. *Philippine Agriculturist* **14**, 289–296 illus.
6 PETRIE G.A. (1988) Races of *Albugo candida* (white rust and staghead) on cultivated Cruciferae in Saskatchewan. *Canadian Journal of Plant Pathology* **10**, 142–150.
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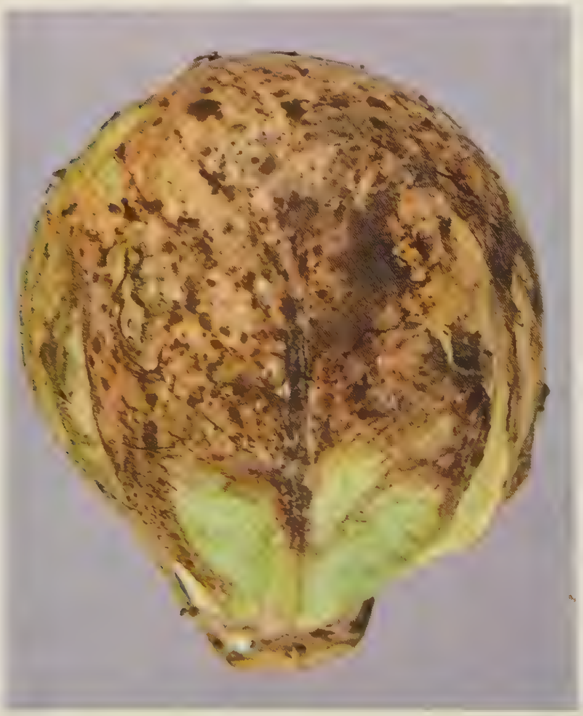
9 STONE J.R., VERMA P.R., DUECK J. & SPURR D.T. (1987) Control of *Albugo candida* race 7 in *Brassica campestris* cv. Torch by foliar, seed, and soil applications of metalaxyl. *Canadian Journal of Plant Pathology* **9**, 137–145.
10 TOGASHI K. & SHIBASHI Y. (1934) Biometrical and biological studies of *Albugo candida* (Pers.) O. Kuntze in connection with its specialization. *Bulletin of the Imperial College of Agriculture and Forestry, Morioka* No. 18, 88 pp illus.

OTHER DISEASES of cabbages, kales, cauliflowers, broccoli, Brussels sprouts and Chinese cabbages

ANTHRACNOSE occurs on Chinese cabbages (and other crucifers) in the USA, and has been attributed to *Colletotrichum higginsianum* Sacc. (9). The fungus exists in the asexual state (acervuli giving rise to conidia), and induces rounded yellowish lesions on leaves and stems. Disease development is favoured by temperatures between 25° and 30°C, but the fungus can also make slight growth at 5°C. Control involves spraying the crop with fungicide and ensuring that plant residues are promptly destroyed.

CERCOSPORA SPOT, caused by *Cercospora brassicae* P. Henn. has been recorded on Chinese cabbages in the Philippines, and the fungus is also capable of infecting other cruciferous crops and weeds (5). The symptoms are pale grey spots, later turning brown with ash-grey centres. Spores (conidia) are dispersed by wind, insects and water, and disease development is favoured by temperatures between 25° and 30°C. Control is by sanitation, rotation and fungicide sprays.

CHOANEPHORA ROT has been reported from India, where the fungus *Choanephora cucurbitarum* (page 27) causes a post-harvest rot of cauliflowers during transit and storage (7). In the field, greyish-brown spots appear on the leaves, which later become covered with a silvery 'bloom' of mould growth. The curd may undergo a soft wet decay.



206 Cladosporium rot of cabbage

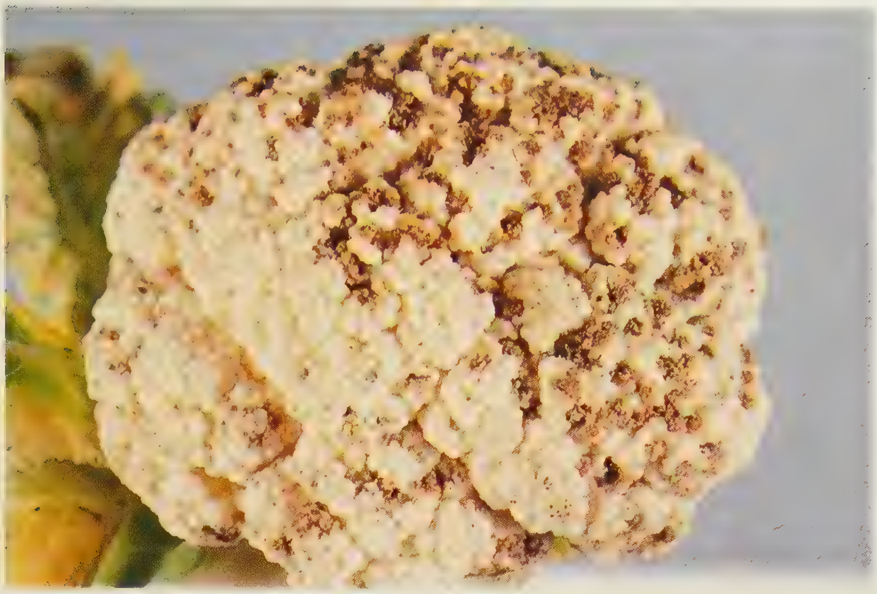
CLADOSPORIUM ROT has been recorded on cabbages, cauliflowers (4), broccoli (6) and Brussels sprouts, and is characterised by the production of a velvety mass of dark olive-green spores. In Taiwan the causal organism has been identified as *Cladosporium tenuissimum*, said to be seed-borne. From the USA (California) there is a report of superficial colonisation of cauliflower curd tissue by a *Cladosporium* sp., necessitating removal by means of a jet spray of chlorinated water, followed by a chlorine dip.

FUSARIUM ROT, caused by *Fusarium roseum* Link emend. Snyder & Hansen, occurs infrequently on stored cabbage in the UK, Canada and the USA (1). A white mould develops, accompanied by a yellow pigment at the surface of infected tissue. Rapid ageing of tissue occurs, and in this respect it resembles the disease called 'yellows' which is caused by *Fusarium oxysporum* f. sp. *conglutinans*. Decay is not held in check by modified atmosphere storage (1). *Fusarium* spp. may also infect cauliflower crops weakened by frost and drought.

MUCOR ROT, caused by species of the fungus *Mucor*, has been recorded on broccoli and Brussels sprouts in the USA (6,10); in modified atmospheres (low oxygen) mucor rot predominated over other types of decay (6).

NIGROSPORA ROT of cauliflowers occurs in Egypt and is attributed to *Nigrospora sphaerica* (Sacc.) Mason (8). Symptoms include brown patches on the curd and, on the stalklets, elongated sunken blackish-brown spots with raised edges.

PYTHIUM ROT of cabbages may be caused by any of several species of *Pythium*, and is characterised by a firm to soft wet decay accompanied by a mild odour resembling that of stewed cabbage. This disease has been recorded in the USA (2) but does not appear to be very common. Like phytophthora rot (q.v.), to which it bears some resemblance, pythium rot is likely to be masked by secondary bacterial soft rot.



207 Fusarium rot of cauliflower

RHIZOPUS ROT has been recorded on cabbages, cauliflowers and Brussels sprouts. A pale brown soft watery rot develops, and under humid conditions a coarse white mould is produced, bearing black spore heads (sporangia). The disease may be caused by *Rhizopus stolonifer* (page 81) or *R. oryzae* (page 373), which have optimal growth temperatures of 20° and 30°C respectively. Decay is negligible at temperatures below 8°C.

SCLEROTIUM ROT, caused by *Corticium rolfsii* (page 255), may occur in warm countries, especially after a wet growing season. The fungus has been recorded on the leaves of cauliflower in India, causing a severe wet rot (7). White mould develops, and also small brown spherical resting bodies (sclerotia). Cool storage inhibits further decay.

TYPHULA ROT, caused by *Typhula brassicae* (Berg. ex Fr.) Vang, has been recorded on stored cabbages in Finland and East Germany (3). The decay is soft and watery; rotting is initially confined to the outermost leaves but eventually (after several months of storage) the entire head may be invaded. Both on its surface and within are to be found numerous small spherical bodies (sclerotia) 0.5 to 2 mm in diameter, pale at first, later brown and finally black.

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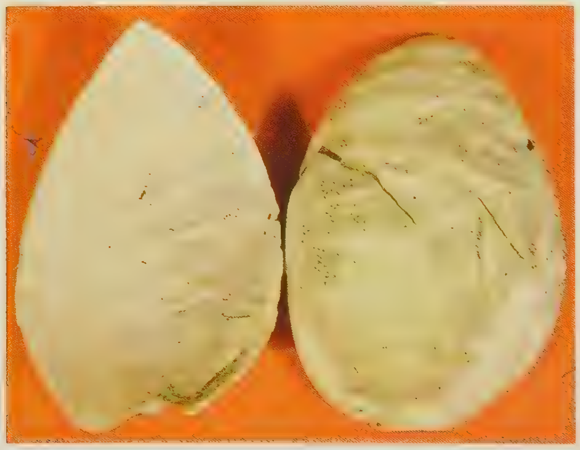
BLACK MID-RIB of cabbages has received particular attention in Canada (2). Discoloration tends to occur towards the base of the mid-ribs, near the butt, though blackening can occur further up the mid-rib if this is ruptured. Although the altered tissue is beneath the surface, it is clearly visible through the epidermis and can be a gross disfigurement. The disorder appears to be genetically determined and occurs only in certain cultivars. In contrast to 'vein-streaking' (q.v.), black mid-rib is aggravated by controlled atmosphere storage (2).

BLACK SPECK of cabbages is also known as **pepper spot**. It is said to be distinct from 'grey speck' (q.v.) (1), although some investigators (3,10) have presumed that all three names are synonymous. Black speck results from discoloration and collapse of tissue surrounding the natural openings (stomata) in both inner and outer leaves of the head. Initially specks are minute, but they may develop further in store, coalescing into more substantial lesions (4). Cultivars differ in susceptibility to black speck, and soil factors are also influential (3). In view of the role played by plant nutrition, suggested means of control have included spraying the growing crop with manganese salts (3). Post-harvest fungicide dips seem to increase susceptibility to the disorder (4,10), whereas controlled atmosphere storage is beneficial (3).



208 Black speck (pepper spot) of cabbage

BLACK SPECK of cauliflowers can be a problem in some North American cultivars which have dense globular curds used in the processing industry (7). The heads may appear perfect externally, but when they are broken apart into individual flower-stalks (e.g. for freezing or pickling), black specks may be found on some of the stalks. Lesions are from 0.5 to 4 mm in diameter. The disorder tends to follow a warm season and rapid growth, during which mineral uptake may be inadequate. It is sometimes associated with internal browning/hollow stem (q.v.) of the main stalk, and may therefore be related to boron deficiency.

GREY SPECK of cabbages is a superficial blemish which tends to occur near the base of the outer leaves (1). Specks on the leaf blade may coalesce to form a reticulate pattern of small spots. The main and lateral veins often show a dark grey shading, easily confused with 'vein-streaking' (q.v.). Susceptible cultivars should be stored in controlled atmosphere, which inhibits symptom development.

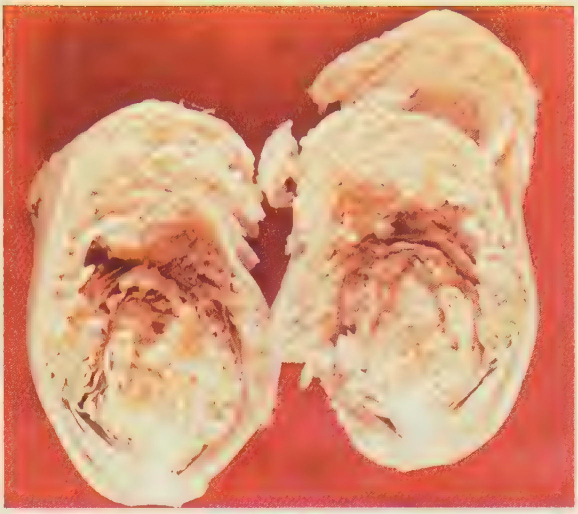
HOLLOW STEM of cauliflower and broccoli develops in the field. Elliptical cracks in the inner stem tissue enlarge and coalesce, causing the stem to become hollow. This reduces the quality of the harvested product, especially since affected tissue is often discoloured. Cultivars differ greatly in susceptibility to this disorder, which is caused by a deficiency of boron (8). Control is achieved by an appropriate fertiliser regime with not too much nitrogen. Over-rapid growth (a predisposing factor) can also be prevented by closer spacing of plants.

INTERNAL BROWNING of Brussels sprouts is caused by a deficiency of calcium and, like tip-burn (q.v.), is especially likely when susceptible cultivars undergo rapid growth. Incidence is highest when the crop is harvested late, following long periods of frosty weather (6). If calcium treatment is ineffective, it is suggested that earlier sowing, planting and harvesting can help to prevent the disorder (6).

NECROTIC SPOT of cabbages is characterised by black sunken lesions larger than 1 mm across, circular to oval or irregular in shape, located on or along the mid-rib and on either side of the leaf blades (2). Dark lesions may also develop within the main stalk tissue. In contrast to 'black speck' (q.v.) but in common with 'black mid-rib' (q.v.), necrotic spot is aggravated by controlled atmosphere storage.

PEPPER SPOT of cabbages is the same as **black speck** (q.v.).

TIP-BURN of cabbages is especially serious when it affects the inner leaves. Their margins become brown and papery, later turning dark brown to black. The affected area may be restricted to the edges or may extend to half the leaf. Symptoms develop in the field, but internal tip-burn is visible only if the head is sectioned or the outer leaves are removed (5). Although the disorder itself does not progress after harvest, the injured tissue is predisposed to attack by soft-rot bacteria. Tip-burn results from a maldistribution of calcium within the plant. Even though ample calcium may be taken up, it is translocated preferentially to the outer leaves, especially if they are undergoing rapid growth. Thus, while calcium sprays can prevent tip-burn of outer leaves, the treatment may have little effect on inner leaves (9). Various strategies have been suggested to minimise the likelihood of internal tip-burn in susceptible cultivars. Closer spacing of plants and reduced application of nitrogen ensure that growth rates are not excessive. Soil moisture should be adequate at all times. The use of wind-breaks and a policy of night-time misting help to minimise transpiration during the hours of darkness. This is beneficial in promoting efficient distribution of calcium within the plant (5); the explanation is similar to that given under 'blossom-end rot of tomatoes' (q.v.) which is likewise a calcium deficiency disorder.



209 Tip-burn of cabbage

VEIN STREAKING of cabbages consists of superficial black or brown markings on the leaves, in the immediate vicinity of the mid-rib. As in 'grey speck' (q.v.), the discoloration may exhibit a reticulate pattern on the mid-rib or it may follow the course of lateral veins (1). More advanced symptoms sometimes include death and discoloration of vascular tissue in the mid-rib and veins; this results in black, thread-like strands within the leaf tissue. Susceptible cultivars should be stored in a controlled atmosphere, since this inhibits development of the disorder (1).

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See also:

BERKEL N. van (1988) Preventing tipburn in chinese cabbage by high relative humidity during the night. *Netherlands Journal of Agricultural Science* **36**, 301–308.

CHAPTER 6

MISCELLANEOUS FLOWER, STEM AND LEAF VEGETABLES

Artichokes

The **globe artichoke**, *Cynara scolymus* L., belongs in the family Compositae, and is native to the Mediterranean region (5). The closely related **cardoon**, *Cynara cardunculus* L., was popular in ancient times, but the globe artichoke is known with certainty only from the Middle Ages (9). It should not be confused with the Jerusalem artichoke (q.v.). Current production of globe artichokes is well over 1 million tonnes, the main producers being Italy, Spain and, to a lesser extent, France. Other growing areas include North Africa, the eastern Mediterranean, California, parts of South America, and New Zealand.

The plant produces large thistle-like flowers, and it is the plump young flower buds which are harvested. Each consists of a fleshy receptacle from which arise thick, overlapping bracts (9); in the centre is a mass of hair-like immature florets comprising the 'choke' (7). The edible parts are the receptacle or base, and the lower fleshy portion of each bract. 'Artichoke hearts' are small buds with the tough outer bracts removed (9).

The artichoke is a perennial crop, which flowers either in summer or in winter, according to cultivar (2). Spraying with a growth-regulating chemical can advance harvest-time by several weeks (2,9). Great care is needed in the post-harvest handling of artichokes. The harvested buds respire at a high rate, and must be pre-cooled promptly and thoroughly; vacuum-cooling (1), hydro-cooling (3) or room-cooling (7) may be used. Optimal holding conditions are a temperature of 0°C and a relative humidity approaching saturation (6,7,10). Such an environment can be achieved by means of top ice during transport to market. Alternatively, if mechanical refrigeration is used, a high humidity can be maintained with plastic film packaging (6,8,10).

The maximal storage life of artichokes is reported to be between 2 and 7 weeks (6,7,9,10). The wide variation probably arises from differences in cultivar characteristics (1), in quality at harvest (9) and in standards used to define post-storage acceptability. It is reported that controlled atmosphere may confer extra benefit (8,9), be of no advantage (7), or cause physiological damage such as blackening of the inner bracts (1,7). A more general blackening may be indicative of senescence or alternatively severe freezing injury, which can occur before or after harvest. Even slight frost damage may be significant; although culinary quality is not impaired, affected buds have a shorter potential storage life as a result of their increased susceptibility to decay (4,9).

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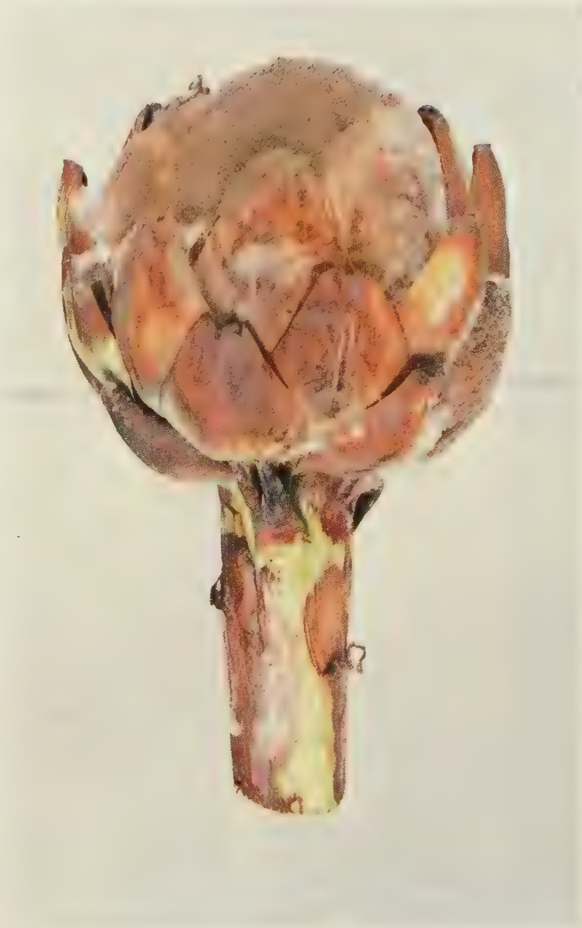
GREY MOULD ROT of artichokes caused by

Botryotinia fuckeliana **(de Bary) Whetzel**
Conidial state: *Botrytis cinerea* Pers.

OCCURRENCE This is the most common and serious disease of artichokes. Records include the USA (2,3,5), Italy (1) and France (4).

SYMPTOMS Reddish-brown to black sunken lesions tend to develop first at the tips of the bracts (2,3), although invasion may also occur via the cut stem (2,4). Subsequently the entire head may decay. In a dry atmosphere the affected tissue remains fairly firm and there is no obvious mould growth. Under moist conditions the lesions have watersoaked borders and support a whitish mould which (if the humidity is subsequently reduced) gives rise to a dense mass of brownish-grey spores (2). Tough dark resting bodies (sclerotia) may also form on decayed tissue (4).

BIOLOGY Sclerotia persist in the soil, and the fungus also survives in plant debris (whether from artichokes, other vegetable crops or weeds) on which it produces quantities of spores (conidia) (2). Spore dispersal is by air currents and, in moist conditions (such as foggy weather), artichoke heads may be infected via the growth cracks which are often present at the tips of the bracts (2,3). Alternatively the heads may simply be contaminated with spores and later become infected via injuries sustained during harvesting and handling (2,3). It has also been noted that artichokes weakened by frost or by attacks of downy mildew (q.v.) are predisposed to grey mould rot during storage and transit (2,4). In a warm humid atmosphere the mould spreads into adjacent healthy heads (2).



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CONTROL Plant debris should always be promptly removed and destroyed (2). In the event of pre-harvest infection, the crop should be sprayed with a suitable fungicide as soon as symptoms are apparent (1). Careful harvesting and handling are important in minimising physical injury and thereby reducing the likelihood of post-harvest infection (2,3). Prompt and rapid cooling is essential; it has been established that refrigeration is more effective in curbing infection than in retarding decay which has already commenced (3). Grey mould continues to develop slowly even at 0°C, and resumes rapid growth on return to ambient temperature, the optimum lying between 22° and 25°C (2).

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1 CORDA P., FIORI M. & CARTA C. (1982) Osservazioni sul "marciume dei capolini" del carciofo (*Cynara scolymus* L.) da *Botrytis cinerea* Pers. in Sardegna. *Studi Sassaresi III* **29,** 449–454.
2 LINK G.K.K., RAMSEY G.B. & BAILEY A.A. (1924) Botrytis rot of the globe artichoke. *Journal of Agricultural Research* **29,** 85–92 illus.
3 LIPTON W.J. & HARVEY J.M. (1960) Decay of artichoke bracts inoculated with spores of *Botrytis cinerea* Fr. at various constant temperatures. *Plant Disease Reporter* **44,** 837–839.
4 MOREAU M. (1961) Une altération des artichauts. *Bulletin of the Research Council of Israel D* **10,** 219–222 illus.
5 TAVERNETTI A.A. (1933) Production of the globe artichoke in California. *Circular of California Agricultural Extension Service* No. 76, 24 pp illus.

WATERY SOFT ROT of artichokes caused by
Sclerotinia sclerotiorum **(Lib.) de Bary**

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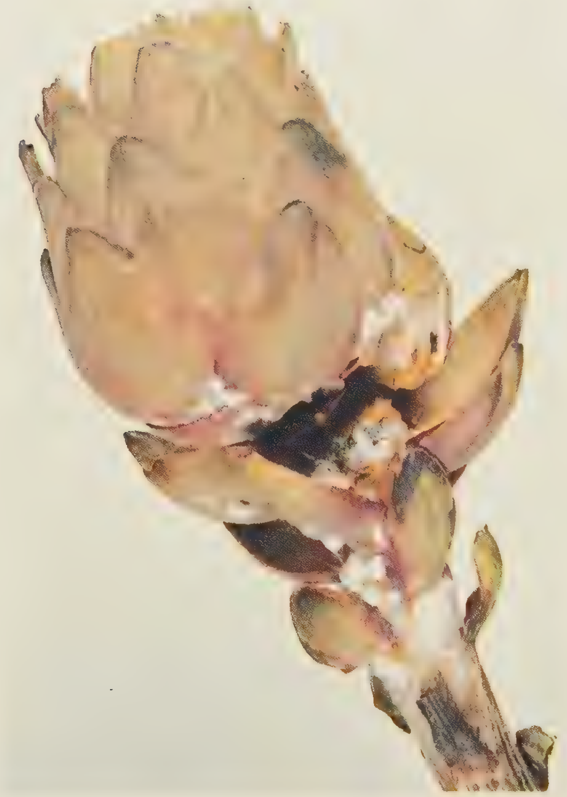

OCCURRENCE The author has observed this fungus in a consignment of globe artichokes imported into the UK from Italy. References in the literature, from Italy and the USA, describe the disease in the field, usually a stem-rot and wilt (2,3,5). The fungus, which attacks many crops, is common in temperate countries and in subtropical regions possessing a cool wet season.

SYMPTOMS Affected bracts are soft and water-soaked and, in a humid atmosphere, lesions become covered with a closely appressed white mould. Irregularly shaped bodies (sclerotia) develop on the mould and within decayed tissue; these are white at first and later black, with dimensions of several mm (2).

BIOLOGY The sclerotia persist in the soil and, following a period of wet weather or frequent irrigation, they characteristically produce the sexual stage (apothecia giving rise to ascospores) (5). In most vegetable crops it is these airborne spores which cause primary infections. Probably the 'heads' of artichokes can be infected in this manner; this would explain the observation (made in New Zealand) that seeds of artichokes may be completely invaded by the fungus, the seed contents being converted into a sclerotium (1). It is conceivable also that airborne spores are washed down the stem by rain or irrigation water and cause infection at soil level, leading to stem-rot and consequent wilting of the plant. Alternatively, stem-rot may result from infection by mould strands growing directly from sclerotia. Both types of behaviour have been reported for *S. sclerotiorum* on sunflower, which sometimes suffers a 'head-rot' and sometimes a stem-rot and wilt (4).

CONTROL In new plantings it is essential to sow clean seed (1,5). Irrigation should be carefully regulated, and general cultural practices should be directed towards producing a well-aerated crop which does not retain water drops for extended periods (5). In the event of infection, diseased plants should be carefully removed and burned, before more sclerotia can develop. Once in the soil, sclerotia are very difficult to destroy; various chemical, physical and biological methods have been described in connection with other crops (watery soft rot, q.v.). Harvested artichokes should be cooled as soon as possible. At temperatures below 5°C decay proceeds slowly, although rapid fungal growth is resumed at ordinary temperatures (5).

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1 COLE A.L.J. (1980) A blind-seed disease of globe artichoke. *Transactions of the British Mycological Society* **74,** 199 illus. [Note].

2 GRASSO V. (1950) Nuove e gravi malattie di piante ortive in Italia. *Rivista della Ortoflorofrutticoltura Italiana* **34,** 16–19 illus.

3 HEALD F.D. (1931) Division of Plant Pathology. *41st Annual Report of Washington Agricultural Experiment Station* (Bulletin No. 260), 47–50.

4 HUANG H.C. (1985) Factors affecting myceliogenic germination of sclerotia of *Sclerotinia sclerotiorum. Phytopathology* **75,** 433–437.

5 MARRAS F. (1962) I "marciume del colletto" del carciofo causati dalla *Sclerotinia sclerotiorum* (Lib.) Massee, dalla *Sclerotium rolfsii* Sacc. e dalla *Rhizoctonia solani* Kühn, in Sardegna. *Studi Sassaresi III* **10,** 90–103 illus.

OTHER DISEASES of artichokes

ASCOCHYTA ROT, caused by *Ascochyta cynarae* Died., has been recorded in Algeria (7), Tunisia, Morocco (8), Italy, France (9) and the UK (3,6). Infection usually begins at the tips of the outermost bracts and, if the growing season is fairly dry, these lesions do not progress beyond a superficial blemish (8). On the other hand, if dew or rain drops persist within the flower head, internal infection can occur, which does not become apparent until decay is well-advanced. A dark wet rot ensues, with copious production of minute black bodies (pycnidia giving rise to conidia) (6,8). The spores are disseminated by water, and may infect all aerial parts of the plant. Cultivars with compact flower heads are more susceptible to head rot than are those with more open heads (8). Control measures include prompt destruction of diseased plants, surface disinfection of soil, and the use of preventive fungicide sprays on the developing crop (8).

BACTERIAL ROT may be caused by *Xanthomonas* sp., which tends to attack artichokes after a frost (9). The bacteria gain entry through the natural openings (stomata) which are most prevalent on the outer surface of the bracts. Dark green oily spots develop, and in a humid atmosphere there is a yellowish exudate of bacteria (9). The lesions coalesce and turn brown, and eventually the entire head may undergo secondary bacterial soft rot. A black rot, caused by *Erwinia* sp., has been reported from Turkey (10). Buds are attacked, and the disease may be serious since chemical control is generally ineffective.

DOWNY MILDEW is caused by *Bremia lactucae* (page 203), which has been recorded on artichokes in Morocco (1), Italy (5), France (9), Czechoslovakia and the UK. Pinkish-white patches of mould may be found on the inner surface of the lowermost bracts, as well as on the leaves. Downy mildew is essentially a field disease, but it predisposes artichokes to ascochyta rot (q.v.) and grey mould rot (q.v.) after harvest.

ITERSONILIA ROT has been recorded on artichokes in the UK (6). Lesions develop first on the uppermost bracts, and coalesce to form extensive dark brown areas which render the heads unmarketable. The causal fungus is *Itersonilia perplexans* (page 283).

POWDERY MILDEW, caused by *Leveillula taurica* (Lev.) Arnaud, has been recorded in the UK (3), Morocco (2), Egypt, Israel, Spain and Italy (4). In countries with a hot dry season the fungus is likely to produce the sexual stage. The spore-producing bodies (cleistothecia) are formed on the undersurface of leaves and subsequently fall on to the soil (2). They can survive from season to season and, under favourable conditions, ascospores are released and cause infection. In more temperate localities the fungus exists solely in the asexual state (conidia), this form being known as *Ovulariopsis cynarae* Ciccarone (4). The artichoke crop becomes increasingly susceptible to infection as it reaches the heading stage.

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1 ANON. (1937) In *Memento du Service de la Défense des Végétaux (Maroc)* No. 44.

2 BERGER G. (1938) Contribution à la connaissance de *Leveillula taurica* Arnaud. *Annales des Épiphyties* NS **4,** 21–25 illus.

3 BROOKS A.V. (1970) Notes from Wisley. Review of plant diseases, 1969. *Journal of the Royal Horticultural Society* **95,** 4–8.

4 CICCARONE A. (1951) La “nebbia” del carciofo (*Cynara scolymus* L.) e del cardo (*Cynara cardunculus* L.). *Bollettino della R. Stazione di Patologia Vegetale, Ser. 3,* **9,** 163–204 illus.

5 CICCARONE A. (1969) Attuali cognizione sulle malattie del carciofo. *Atti I Congreso Internazionale degli Studi sul Carciofo,* 181–192.

6 CRAGG I.A. (1966) New or uncommon plant diseases and pests. *Itersonilia perplexans* on globe artichoke. *Plant Pathology* **15,** 47 illus.

7 KILLIAN C. & MAIRE R. (1928) Sur une nouvelle maladie des artichauts et sur un champignon *Diplodina cynarae* qui l’accompagne. *Bulletin de la Société d’Histoire Naturelle de l’Afrique du Nord* **19,** 20–23 illus.

8 MALENÇON G. (1936) Une grave maladie des artichauts au Maroc. *Revue de Mycologie NS* **1,** 165–175 illus.

9 RIDÉ M. (1956) Sur une maladie nouvelle de l’artichaut (*Cynara scolymus* L.). *Comptes Rendus hebdomadaires des Séances de l’Académie des Sciences, Paris* No. 243, 174–177.

10 TÜRKMENOĞLU Z., GÜNDOĞDU M. & KAYA S. (1975) [Black rot (Erwinia) of artichoke buds in Turkey.] *Bitki Koruma Bülteni* **15,** 43–57.

Asparagus

A member of the family Liliaceae, *Asparagus officinalis* L. is native to Europe and eastern Asia, and may still be gathered from the wild. The crop is cultivated on a commercial scale in the USA (4,8), Canada (7), South America (e.g. Chile), Australia (10), New Zealand (2,6), Taiwan, Japan (5) and most European countries (1).

It is the young tender shoots or 'spears' which are harvested, and they fetch a high price in the market. The spears are green or purplish, unless they have been blanched by 'earthing up' at the time of emergence. Spear development is rapid, energy being derived from food reserves in the fleshy underground stem or 'crown'. After the spears have been harvested, the plants are encouraged to produce vigorous fern-like growth which (by photosynthesis) serves to replenish crown and root reserves. This is necessary if yields are to be maintained.

Harvested spears have a high respiration rate, although it declines within a day or so (6,8). Storage life is short, and may be terminated by disease (principally bacterial soft rot, q.v.) (3,9) or by undesirable physiological changes, notably flavour loss and toughening (q.v.) (4,6). Fibre development is influenced by several factors, including pre-harvest temperature, storage temperature, and length of spear (4). Long thin spears tend to toughen readily, especially if harvested after cold weather and held at a moderate temperature (4). On the other hand, short (7 cm) thick spears have a higher respiration rate and lose their nutritive value more rapidly (8).

Prompt temperature reduction is essential; vacuum cooling is feasible (1) but hydro-cooling in iced water is more usual (1,6,10). The water must be chlorinated in order to control decay organisms (10). As a further precaution against bacterial invasion of the cut ends, the butts can be dipped in a shallow bath of concentrated hypochlorite solution (10). They may subsequently be placed in contact with a moistened pad, which helps to prevent wilting. Harvested spears with access to moisture continue to elongate, by as much as 1 cm or more per day at ambient temperature (4). Furthermore, if the spears are placed in a horizontal position, the tips (being negatively geotropic) tend to bend upwards (10). These properties dictate the design of package appropriate for asparagus; the spears are packed upright, and a 'head space' is left between the tips and the roof of the pack.

The use of plastic film packaging is beneficial in minimising moisture loss (5). Careful choice of film permits the establishment of an in-package modified atmosphere; the increased carbon dioxide level not only reduces respiration rate but also helps to maintain an attractive green colour and inhibits fibre development (10) and decay (1). An inappropriate atmosphere can, however, result in pitting (q.v.), and an alternative method of delaying senescence is to include an ethylene absorbent in the pack (5).

Despite a report of chilling injury (q.v.) at temperatures below 2°C (8), the usual practice is to hold asparagus at 0° to 1°C, the maximal storage time being about 3 weeks (1,5,6,10). Fumigation (against insect pests) reduces storage life of asparagus significantly, by stimulating respiration and ethylene production (2).

1 ANDRÉ P., BURET M., CHAMBROY Y., DAUPLÉ P., FLANZY C. & PELISSÉ C. (1980) Essais de conservation de turions d'asperge au moyen de la préréfrigération par le vide, associée à des atmosphères modifiées et au froid. *Revue Horticole* No. 205, 19–25.

2 BEEVER D.J., YEARSLEY C.W. & HOGG M.G. (1985) Effect of post-harvest fumigation on quality of asparagus. *New Zealand Journal of Agricultural Research* **28,** 537–543.

3 CEPONIS M.J., CAPPELLINI R.A. & LIGHTNER G.W. (1988) Disorders in asparagus, eggplant, and snap bean shipments to the New York market, 1972–1985. *Plant Disease* **72,** 178–182.

4 CLORE W.J., CARTER G.H. & DRAKE S.R. (1976) Pre-harvest and post-harvest factors affecting textural quality of fresh asparagus. *Journal of the American Society for Horticultural Science* **101,** 576–578.

5 ITOH K. (1986) [Storage quality of fruits and vegetables. 1. Storage of green asparagus.] *Memoirs of the Faculty of Agriculture, Hokkaido University* **15,** 7–18.

6 KING G.A., HENDERSON K.G., O'DONOGHUE E.M., MARTIN W. & LILL R.E. (1988) Flavour and metabolic changes in asparagus during storage. *Scientia Horticulturae* **36,** 183–190.

7 LOUGHEED E.C. & TIESSEN H. (Eds.) (1985) *Proceedings of the 6th International Asparagus Symposium.* University of Guelph, Canada, 408 pp.

8 SALTVEIT M.E. & KASMIRE R.F. (1985) Changes in respiration and composition of different length asparagus spears during storage. *HortScience* **20,** 1114–1116.

9 SMITH M.A., McCOLLOCH L.P. & FRIEDMAN B.A. (1966) Market diseases of asparagus, onions, beans, peas, carrots, celery, and related vegetables. *Agriculture Handbook of the United States Department of Agriculture* No. 303, 65 pp illus.

10 TOMKINS R.B. & CUMMING B.A. (1988) Effect of pre-packaging on asparagus quality after simulated transportation and marketing. *Scientia Horticulturae* **36,** 25–35.

BACTERIAL SOFT ROTS of asparagus caused by
***Erwinia* spp.**
***Pseudomonas* sp.**

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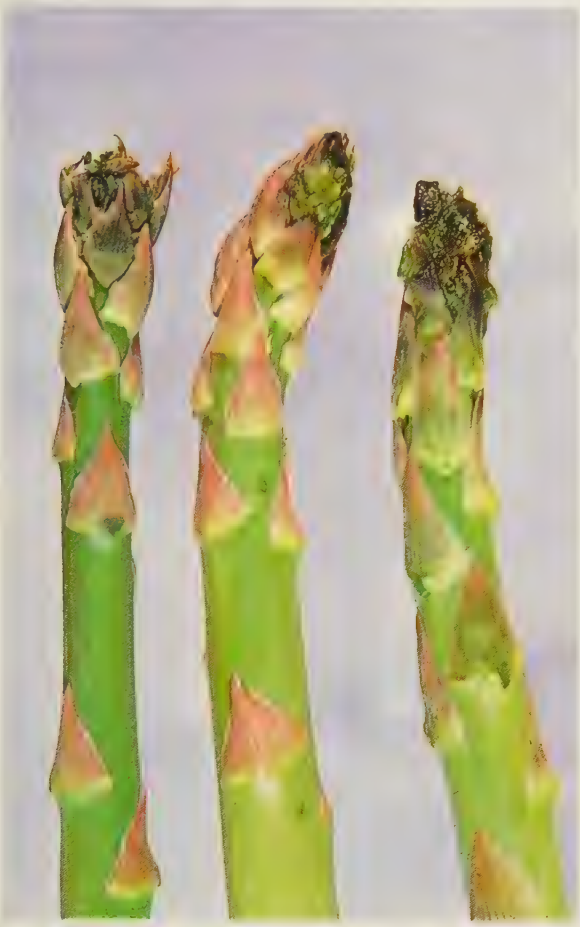

OCCURRENCE Bacterial soft rot is the commonest post-harvest disease of asparagus and can cause serious losses (4). Species of *Erwinia* have been recorded in the USA (1,4) and *Pseudomonas* sp. in the USA (1) and in New Zealand (2).

SYMPTOMS Decay may occur anywhere on the spears, in the form of 'soft rot pits' (2). Frequently, however, it is the butt ends or the tips which are affected (4). In the early stages of infection the tips may appear almost normal externally but, if pressure is applied, drops of liquid can be expelled, containing millions of bacteria. Diseased tissue is soft, water-soaked, dark green and slimy, with a disagreeable odour (4).

BIOLOGY These bacteria are common soil inhabitants (1) and, since soil particles can be blown about by wind or be splashed up by rain, the harvested spears are likely to be contaminated. Infection occurs in the presence of moisture, via wounds sustained before, during or after harvest (1). Once established, the bacteria can cause secondary infection of undamaged spears in contact. The rate of decay is determined by the storage environment, being extremely rapid at temperatures above 15°C and somewhat inhibited below 5°C, depending on the species (1,2,4).

CONTROL Careful handling is essential in order to minimise injury. Rapid reduction of temperature is important and, if hydro-cooling (5) is used, the water should be properly disinfected (1) and frequently changed. Only sound spears with tight tips should be packed, since loose or 'feathered' tips are especially prone to bacterial soft rot (3). Exposure to an atmosphere with increased carbon dioxide, either as a pre-storage treatment or as a storage regime, can provide partial control of bacterial soft rot; for reasons which are not understood, a high carbon dioxide concentration inhibits bacterial rotting at the butt end more effectively than at the tips (3). Efficient refrigeration helps to check decay, but rotting resumes at ordinary temperatures (1,2,4).

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1 CAPPELLINI R.A. (1960) Post-harvest quality control of asparagus. *Horticultural News* **41,** 4060–4061.

2 LILL R.E. (1980) Storage of fresh asparagus. *New Zealand Journal of Experimental Agriculture* **8,** 163–167.

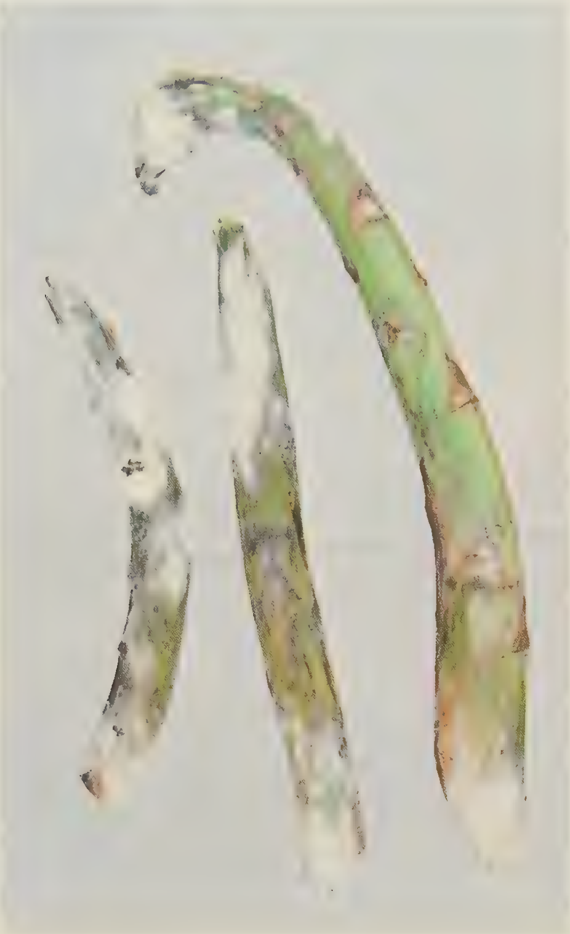
3 LIPTON W.J. (1968) Market quality of asparagus – effects of maturity at harvest, and of high carbon dioxide atmospheres during simulated transit. *Marketing Research Report of the United States Department of Agriculture* No. 817, 7 pp illus.

4 SMITH M.A., McCOLLOCH L.P. & FRIEDMAN B.A. (1966) Asparagus bacterial soft rot – *Erwinia carotovora* (L.R. Jones) Holland. In: *Agriculture Handbook of the United States Department of Agriculture* No. 303, pp 2–3 illus.

5 STEWART J.K. & BARGER W.R. (1961) Effects of cooling method on the quality of asparagus and cauliflower. *Proceedings of the American Society for Horticultural Science* **78,** 295–301.

FUSARIUM ROT of asparagus caused by ***Fusarium* spp.**

213



OCCURRENCE *F. avenaceum* and *F. culmorum* have been recorded on asparagus in New Zealand (4), *F. moniliforme* and *F. oxysporum* f. sp. *asparagi* in the USA (2), Taiwan (5), Italy (3) and the Netherlands (1).

SYMPTOMS Small brown oval lesions may be present at the base of the spears (1) or on the tips or bracts. As the lesions enlarge, a soft rot ensues (4). Under humid conditions a white or pinkish mould develops, producing abundant spores (conidia).

BIOLOGY These fungi are common in soil, where they survive in crop debris or as thick-walled resting spores (chlamydospores). They may also be transmitted in asparagus seed (3). Some cause wilting, foot rot or decline of the growing crop (1,5). Stems are predisposed to infection after damage by insect larvae (2), and a red-brown discoloration around the margins of the damaged areas is typical of fusarium infection. Spears contaminated with spores at harvest may become infected during handling or hydro-cooling (4). At ordinary temperatures decay spreads rapidly into adjacent spears.

CONTROL A fungicide seed treatment may be advisable for new plantings (3). Chemical sprays may be required to control the disease in the field, perhaps by first controlling insect pests (2). Post-harvest rotting is minimised by disinfection of hydro-cooling water (4) and maintenance of low temperature during storage.

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1 BAKEL J.M.M. van & KERSTENS J.J.A. (1970) Foot rot in asparagus caused by *Fusarium oxysporum* f. sp. *asparagi. Netherlands Journal of Plant Pathology* **76,** 320–325 illus.

2 DAMICONE J.P., MANNING W.J. & FERRO D.N. (1987) Influence of management practices on severity of stem and crown rot, incidence of asparagus miner, and yield of asparagus grown from transplants. *Plant Disease* **71,** 81–84.

3 FANTINO M.G. & PAVESI C. (1984) Infezione di *Fusaria* su asparago. *Informatore Fitopatologico* **34**(5), 15–18 illus.

4 LILL R.E. & LAUNDON G.F. (1984) Chlorination of asparagus hydrocooling water for the control of post-harvest decay organisms. *New Zealand Journal of Experimental Agriculture* **12,** 43–45.

5 TU C.C. & CHANG K.F. (1977) [Studies on fusarial wilt and root disease complexes of asparagus in Taiwan.] *Journal of Agricultural Research of China* **26,** 216–223.

See also:

STEPHENS C.T., VRIES R.M. de & SINK K.C. (1989) Evaluation of *Asparagus* species for resistance to *Fusarium oxysporum* f. sp. *asparagi* and *F. moniliforme. HortScience* **24,** 365–368.

PHYTOPHTHORA ROT of asparagus caused by ***Phytophthora*** **spp.**

OCCURRENCE This disease is of major importance, and may be caused by any of several species (5). *P. cactorum* (page 38) occurs on asparagus in France, while *P. cryptogea* Pethybr. & Lafferty is sometimes associated with the disease in California. *P. richardiae* Buisman and *P. megasperma* Drechsler occur on asparagus in Australia. Of greatest importance in New Zealand and California is *P. megasperma* var. *sojae* Hildebrand, and most of the research has been carried out on this species (1,2,3,4,5).

SYMPTOMS The characteristic symptom is an odourless decay accompanied by white mould growth. Rotting may be predominantly at the base of the spears. If the tips become infected, secondary bacteria often follow, inhibiting mould development and imparting a disagreeable odour to the affected tissue (1).

BIOLOGY These fungi are soil-inhabitants, surviving in the sexual state (thick-walled oospores) (5). In wet conditions they produce the asexual state (sporangia, which may give rise to zoospores) (1). Infection occurs after heavy or prolonged rain, or may be associated with the practice of flooding the fields to induce early development of the crop (1). Roots and crowns may be invaded, causing substantial reduction in yield, or even death of the plant (2,3,5). Infected spears show watersoaked, slightly sunken lesions at or just above soil-level, and are usually crooked (3). If infected spears are hydrocooled after harvest, the water becomes contaminated and this can result in infection of the tips also (1).

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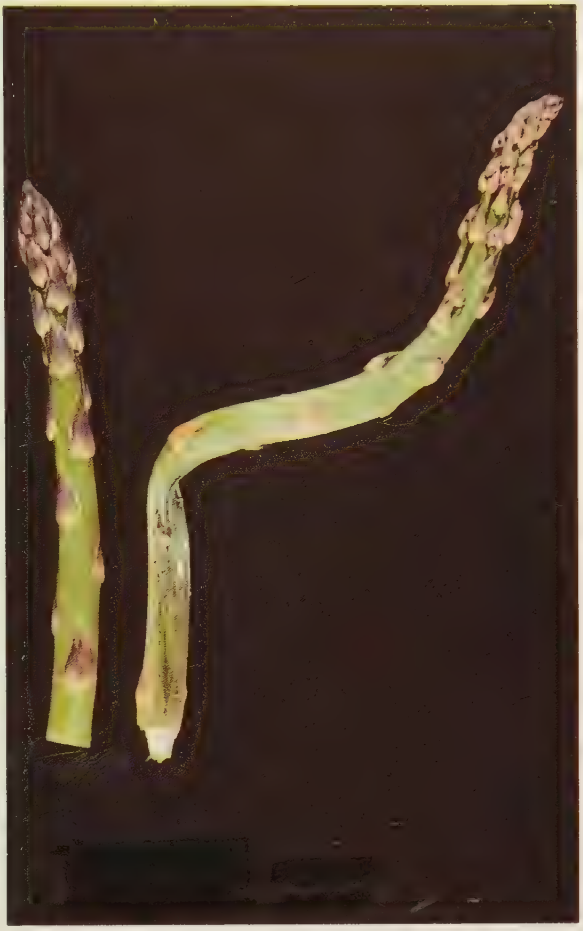

CONTROL The disease can be controlled by means of a single well-timed spray application of an appropriate fungicide (3,4). Cultural methods include improvements in field drainage, the use of seedling transplants instead of crowns, or, alternatively, the storage of crown transplants until field conditions are dry and warm (4). Chlorination of hydro-cooling water helps to protect harvested spears (1). Some asparagus cultivars are partially resistant to this disease (4).

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1 ARK P.A. & BARRETT J.T. (1938) Phytophthora rot of asparagus in California. *Phytopathology* **28,** 754–756 illus.

2 BOESEWINKEL H.J. (1974) Phytophthora on asparagus in New Zealand. *Plant Disease Reporter* **58,** 525–529 illus.

3 CHEAH L.H. (1987) Metalaxyl controls Phytophthora rot of asparagus. *Proceedings of the 40th New Zealand Weed and Pest Control Conference*, pp. 64–66.

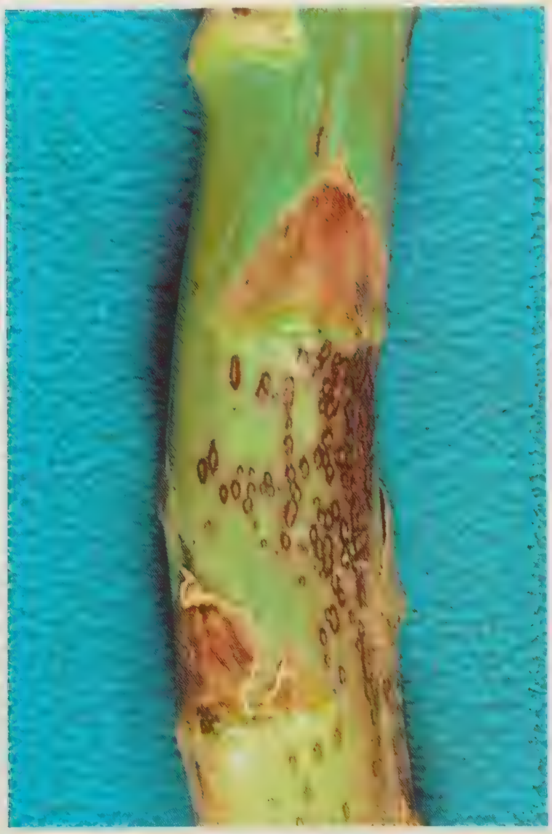
4 FALLOON P.G. (1986) Phytophthora rot of asparagus. 2. Resistant varieties and recommendations for control in established asparagus. *New Zealand Commercial Grower* **41**(9), 23 illus.

5 FALLOON P.G. & GROGAN R.G. (1988) Isolation, distribution, pathogenicity, and identification of *Phytophthora* spp. on asparagus in California. *Plant Disease* **72,** 495–497.

PURPLE SPOT of asparagus caused by

Pleospora allii **(Klotzsch) Ces. & de Not.**
Conidial state: *Stemphylium vesicarium* (Wallr.) Simmons
Pleospora herbarum **(Pers.) Rabenh. ex Ces. & de Not.**
Conidial state: *Stemphylium herbarum* Simmons

215



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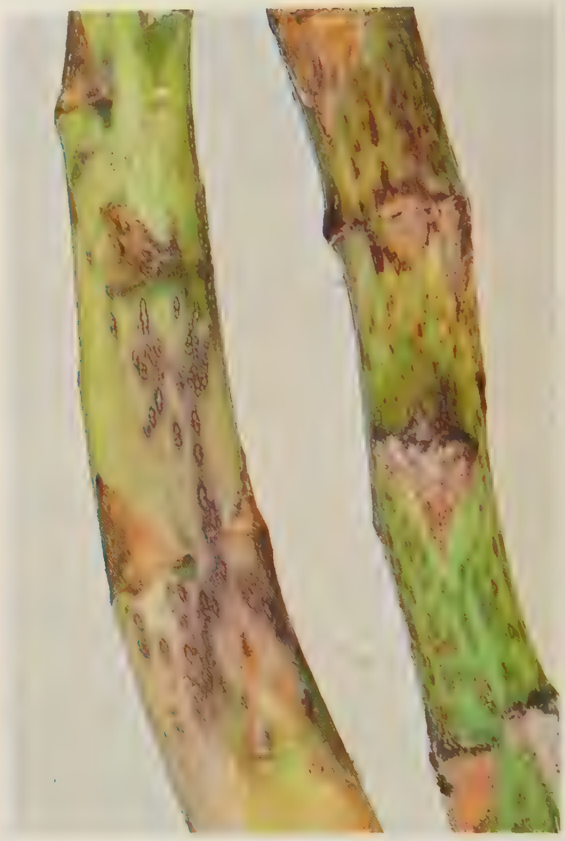
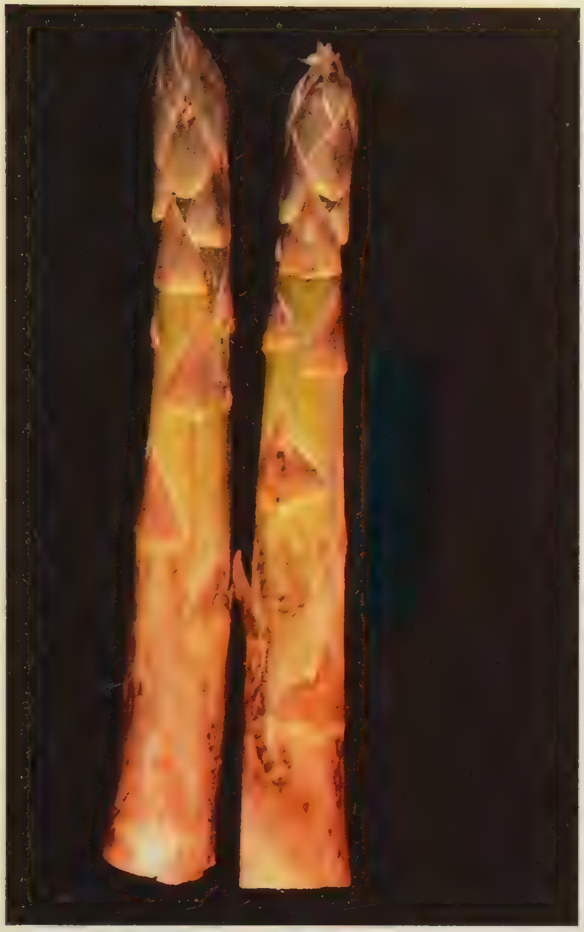


OCCURRENCE Records of the occurrence of purple spot include New Zealand (3) and the USA (4,5,8,9). Leaf spotting, caused by one or other of these fungi, has been reported from Japan (10), New Zealand (1), the USA (5) and several countries in Europe (2,6).

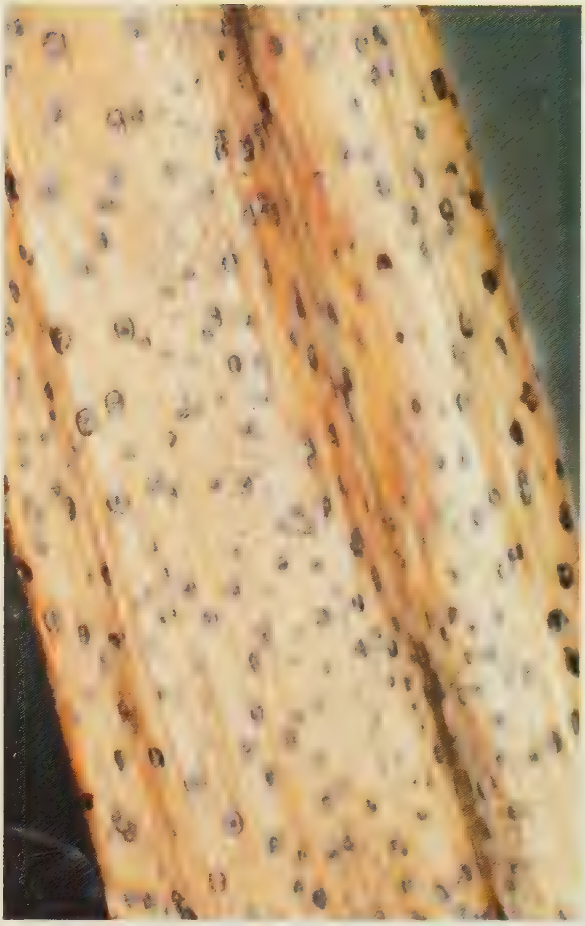
SYMPTOMS The characteristic reddish-purple lesions are small (1 to 2 mm), elliptical, slightly sunken, and usually occur in large numbers on only one side of the spear (9). There is no major breakdown of tissue, but the blemishes are unsightly and detract from market value (9). Processors may reject consignments having more than 3 spots per spear (3).

BIOLOGY The fungi can survive the winter on the surface of asparagus debris, as minute (0.5 mm) black bodies (pseudothecia) (4,5). These give rise to ascospores which are dispersed by wind. Asexual spores (conidia) may also be present on debris or in soil, and either type of spore can cause infection of asparagus spears via the stomata (5). Moisture, whether from rain, irrigation water or dew, is a prerequisite, and injuries predispose the tissue to infection (8). The distribution of lesions on affected spears suggests that a common mode of infection is via wounds made by wind-blown sand (9). Later in the season, lesions may appear on stems and leaves (1,5,6,10), and there is a Canadian report on the occurrence of one of these fungi in asparagus seed (7).

CONTROL In the USA the first appearance of this disease has been linked with the introduction of a 'no-till cultural system' in which the previous year's fern growth is chopped and left on the soil surface (9). If purple spot becomes a problem, it would be preferable to remove and destroy the debris, which may be harbouring the fungus (3,5). Work is in progress to breed resistant cultivars of asparagus (1), and meanwhile the disease is controlled by means of fungicide sprays (3).



217 Purple spot symptoms on freshly harvested spears



218 Pseudothecia in dried asparagus stalk debris

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1 BANSAL R.K., MENZIES S.A. & BROADHURST P.G. (1988) Evaluation of *Asparagus officinalis* L. germplasm for resistance to *Stemphylium* leaf spot. *New Zealand Journal of Experimental Agriculture* **16,** 295–297.

2 BLANCARD D., PIQUEMAL J.-P. & GINDRAT D. (1984) La stemphyliose de l'asperge. *Revue Horticole* No. 248, 27–30 illus.

3 CHEAH L.H. & HUNT A.W. (1987) Stemphylium purple spot of asparagus. *Proceedings of the 40th New Zealand Weed and Pest Control Conference,* pp. 78–79.

4 EVANS T.A. & STEPHENS C.T. (1984) First report in Michigan of the teleomorph of *Stemphylium vesicarium,* causal agent of purple spot of asparagus. *Plant Disease* **68,** 1099 [Note].

5 FALLOON P.G., FALLOON L.M. & GROGAN R.G. (1987) Etiology and epidemiology of Stemphylium leaf spot and purple spot of asparagus in California. *Phytopathology* **77,** 407–413 illus.

6 GINDRAT D., VARADY C. & NEURY G. (1984) Asperge: une nouvelle maladie du feuillage, provoquée par un *Stemphylium. Revue Suisse de Viticulture, d'Arboriculture et d'Horticulture* **16**(2), 81–82, 83–85.

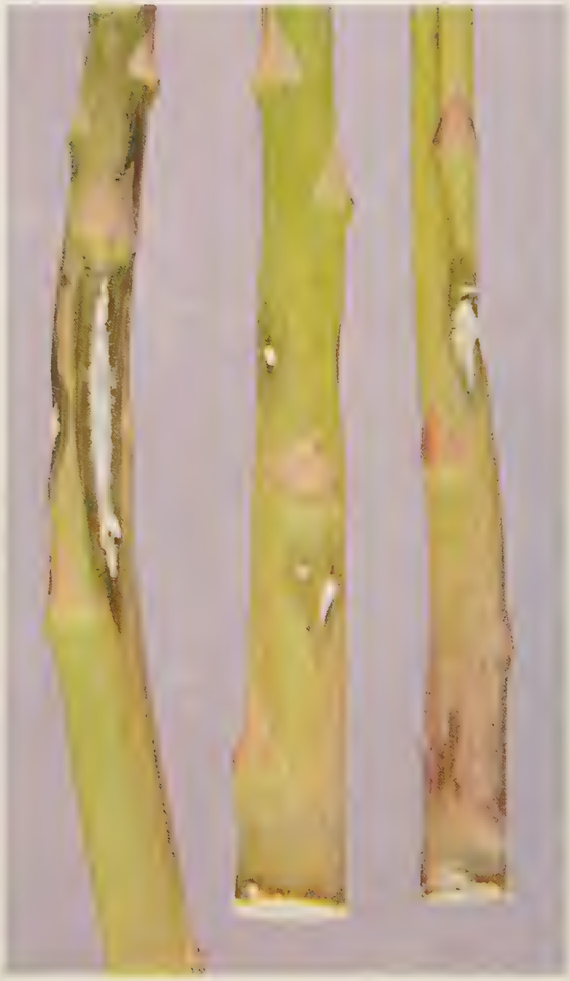
7 GROVES J.W. & SKOLKO A.J. (1944) Notes on seed-borne fungi. 1. Stemphylium. *Canadian Journal of Research C* **22,** 190–199.

8 JOHNSON D.A. & LUNDEN J.D. (1986) Effects of wounding and wetting duration on infection of asparagus by *Stemphylium vesicarium. Plant Disease* **70,** 419–420.

9 LACY M.L. (1982) Purple spot: a new disease of young asparagus spears caused by *Stemphylium vesicarium. Plant Disease* **66,** 1198–1200 illus.

10 SUZUI T. (1973) [Stemphylium leaf spot (*Stemphylium botryosum* Wallr.) on asparagus plants.] *Annals of the Phytopathological Society of Japan* **39,** 364–366.

OTHER DISEASES of asparagus



219 Blue mould rot

BLUE MOULD ROT, caused by *Penicillium* sp., has been recorded as a minor disease of asparagus in France (2). In contrast, it was the principal form of decay in shipments imported into the USA from Argentina (5); serious rotting had occurred both at the butt ends and at bud scales along the sides of the spears. In the USA, where blue mould rot has often been recorded in harvested spears (1), *P. martensii* Biourge (a member of the *P. cyclopium* group) was identified as the cause of a serious crown-rot and root-rot. Recommendations included careful storage of crowns (to prevent their drying out) and hand-sorting before planting, so that any obviously mouldy pieces could be culled and destroyed (1). Spears with incipient blue mould infection benefit from prompt post-harvest cooling in chlorinated water.

GREY MOULD ROT, caused by *Botrytis cinerea* (page 281), has been recorded on asparagus in New Zealand, where the main control measure is attention to hygiene in the packing house and disinfection of water used for hydro-cooling. Other references describe a die-back in the field (3).

PHOMOPSIS ROT is caused by *Phomopsis asparagi* (Sacc.) Bubak, which is possibly synonymous with *Phoma asparagi* Sacc. The disease is serious in Taiwan and India (4). Lesions are small and numerous, light brown with dark borders. The disease is usually described as a stem-blight; it is possible that harvested spears may be affected, though sometimes the plants are killed outright (4).

WATERY SOFT ROT, caused by a species of *Sclerotinia*, has been recorded in the USA and in France (2). *S. minor* attacks asparagus in Germany. Post-harvest rotting is accompanied by a white mould growth on which develop characteristic resting bodies (sclerotia) (page 47).

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1 MENZIES J.D. (1955) A Penicillium crown-rot of asparagus. *Phytopathology* **45**, 527–530.

2 MOLOT P. (1962) Les maladies cryptogamiques de l'asperge. Reprinted from *Revue de Zoologie Agricole et Appliquée* 1962 (4–6), 6 pp.

3 RICH A.E. (1955) Die-back of asparagus associated with *Botrytis cinerea* in New Hampshire. *Plant Disease Reporter* **39**, 305–306.

4 SOHI H.S., ULLASA B.A. & SOKHI S.S. (1975) Stem blight of *Asparagus officinalis* caused by *Phomopsis asparagi* (Sacc.) Bubak. *Current Science* **44**, 722–723.

5 WIANT J.S. & BRATLEY C.O. (1938) Diseases of fruit and vegetables on the New York market during January, February, March and April, 1938. *Plant Disease Reporter* **22**, 190–193.

DISORDERS of asparagus

BENDING can occur within a day or so if harvested spears are kept horizontal and if the temperature is above about 3°C (5). It is for this reason that packages are designed to hold asparagus in the upright position.

CHILLING INJURY is reported to occur after a week or so at temperatures below about 2°C, especially in modified atmosphere storage (4). The tips of affected spears appear limp and dull, the symptoms developing within a few days of removal to a moderate temperature. Chilling injury is not inevitable, however, and there are other reports of successful storage of asparagus at 0° to 1°C for up to 3 weeks.

ELONGATION of spears occurs if the butts are in contact with water and if the temperature is above about 5°C. Tips which make contact with the roof of the package are deflected, with consequent injury. Such problems are controlled by maintaining a sufficiently low temperature during shipment and marketing (5).

FEATHERING is a term applied to the tips of the spears, denoting that the bracts have opened out as a result of growth of the underlying buds. Feathering is a sign of senescence, indicating either that the spears were over-mature when harvested, or that they have been exposed to temperatures higher than optimal after harvest. Besides being unsightly, feathered tips show an increased tendency to decay.

PITTING can result from exposure to an excessive concentration of carbon dioxide, such as may occur within unperforated plastic film packaging in warm conditions (4). Substantial depressed areas are characteristic of injury caused through insufficient oxygen. Complete lack of oxygen results in water-soaking in the zone below the tip, followed by the exudation of amber-coloured liquid (4). Clearly, if an enhanced carbon dioxide concentration is to be used for prolonging storage life, accurate control of atmospheric composition is essential if harmful levels are to be avoided.

TIP ROT, despite its name, is presently classified as a disorder rather than a disease, since no organism is consistently associated with it and no chemical controls it. The longer the storage time at 0°C, the more rapidly tip rot develops during marketing. Deterioration of the tips represents a serious loss in quality, and this disorder has caused major problems in asparagus exported to distant markets (1). The cause is unknown; to date, it has been established that symptoms develop more slowly if spears are held with their butts in water rather than in air, but that soil moisture during growth is not influential (1).

TOUGHENING results from lignification and fibre development (2). These deleterious changes occur progressively from butt to tip, and significant quality loss can occur within a few hours at high ambient temperature. Toughening is accelerated by the presence of ethylene, whether from an external source or from the asparagus itself. The act of harvesting induces the evolution of 'wound ethylene'; moreover, spears cut several cm above ground-level emit more ethylene and toughen more readily than do spears cut at ground level (2). The reason why toughening is retarded by controlled atmosphere storage is that ethylene production is inhibited by high concentrations of carbon dioxide (2).

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1 CARPENTER A., GRBAVAC N., STOCKER A., MESPEL G.J. van der & MARSHALL A. (1988) Studies on asparagus tip rot. *Proceedings of the 41st New Zealand Weed and Pest Control Conference,* pp. 90–95.

2 HAARD N.F., SHARMA S.C., WOLFE R. & FRENKEL C. (1974) Ethylene induced isoperoxidase changes during fiber formation in postharvest asparagus. *Journal of Food Science* **39,** 452–456.

3 KING G.A., HENDERSON K.G. & LILL R.E. (1987) Sensory analysis of stored asparagus. *Scientia Horticulturae* **31,** 11–16.

4 LIPTON W. (1965) Post-harvest responses of asparagus spears to high carbon dioxide and low oxygen atmospheres. *Proceedings of the American Society for Horticultural Science* **86,** 347–356 illus.

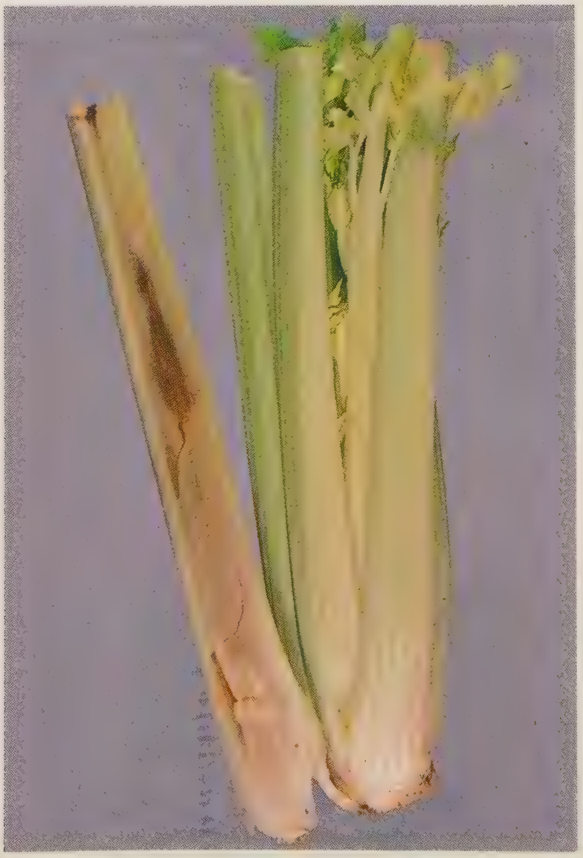
5 MORRIS L.L. & WATADA A.E. (1960) Elongation and bending of asparagus spears. *California Agriculture* **14**(11), 15.

See also:

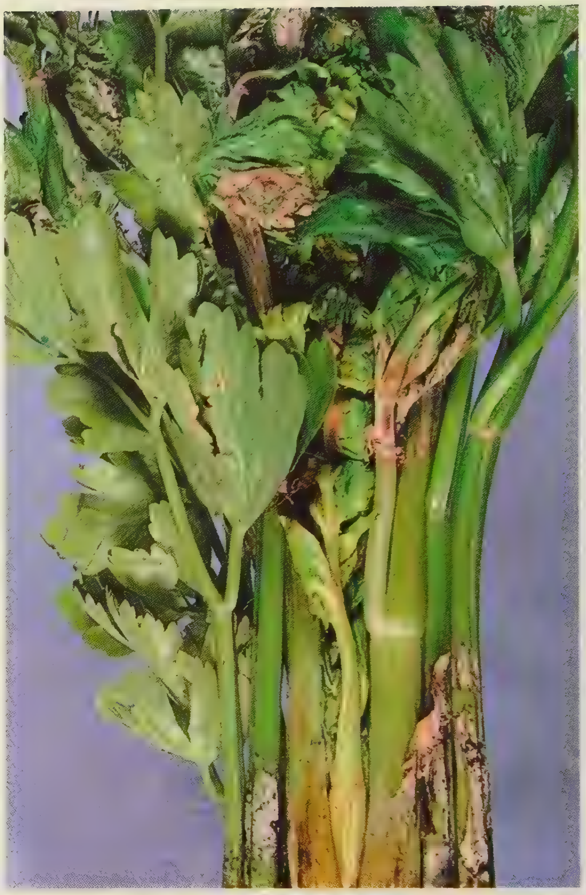
SALTVEIT M.E. (1988) Postharvest glyphosate application reduces toughening, fiber content, and lignification of stored asparagus spears. *Journal of the American Society for Horticultural Science* **113,** 569–572.

Celery and fennel

Celery, *Apium graveolens* L., belongs in the family Umbelliferae and is therefore closely related to carrot and parsley. Celery foliage was used by the ancient Greeks for the making of wreaths, and the plant was also valued for its medicinal properties. Similar leafy types (now designated *A. graveolens* var. *secalinum*) are grown in Mediterranean countries and sold in small bunches for use as a flavouring. Only in recent centuries was there selection for succulent leaf stalks (petioles), which may be eaten raw or cooked. This is *A. graveolens* var. *dulce*, and there are two types. The so-called self-blanching varieties are pale golden yellow, and are popular in the Netherlands, Belgium, France and Spain. Green varieties are preferred in the USA, Israel and the UK; their leaf stalks may be blanched by 'earthing up' during growth, but they are often marketed green. In the UK there is an expanding area given over to early production of self-blanching celery under glass or plastic (8). The problem of 'bolting' is overcome by the nightly provision of a 4-hour period of artificial light, which suppresses flower initiation (8).



220 Bacterial soft rot of celery

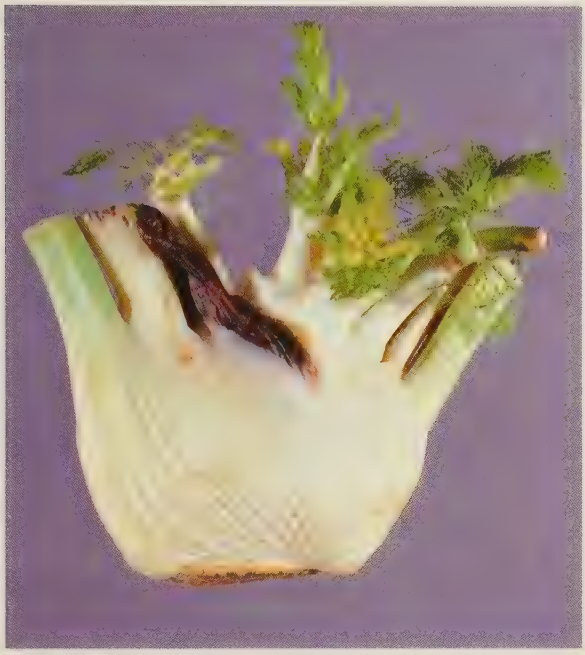


221 *Thanatephorus cucumeris* on celery

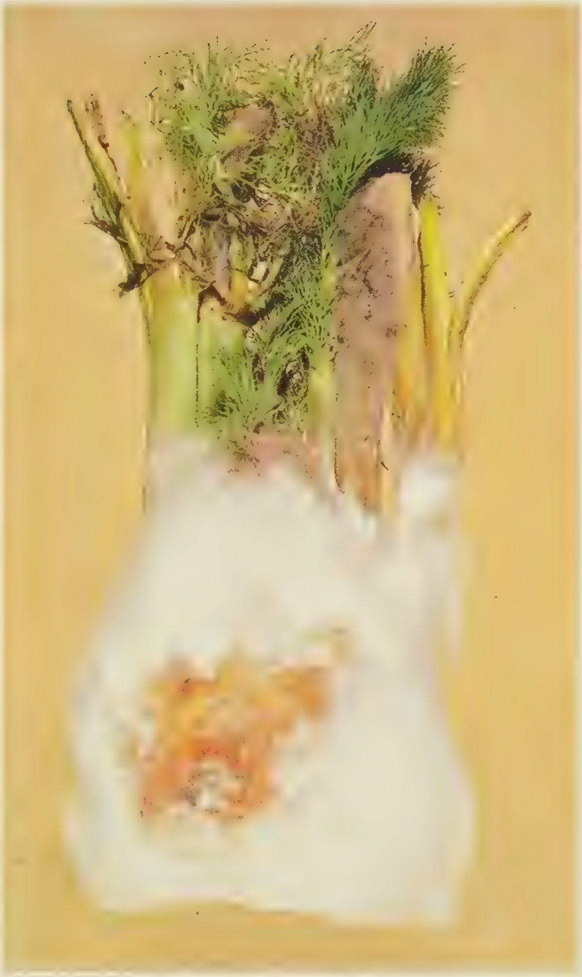
The harvested petioles are sometimes known as celery 'sticks' (8), most of the foliage being removed at the time of lifting. Optimal storage conditions are a temperature of 0°C and a relative humidity close to saturation (7). Prompt and rapid cooling helps to minimise moisture loss; vacuum cooling is feasible but hydro-cooling and forced-air cooling are more usual methods (4,10). Plastic film packaging is beneficial; crate or carton liners should be perforated, and individual polyethylene sleeves are left open at the top.

Celery can be kept for about a month under optimal conditions, longer if it can be re-trimmed before sale. The central stalks elongate, and some blanching is likely during storage in the dark. Major diseases (1,2,3,5,8) are bacterial soft rot, grey mould rot and watery soft rot (q.v.), which are not fully controlled by cold storage. Certain controlled atmosphere regimes have an inhibitory effect on decay organisms, permitting storage beyond 2 months (9). Disorders of celery include blackheart, bruising, and inadvertent freezing (1), the freezing point of celery being only very slightly below 0°C. Even under optimal storage conditions, discoloration of the butt is inevitable.

Florence fennel, *Foeniculum vulgare* Miller, is also known by its Italian name of finocchio (6). It is in the same family as celery. In Florence fennel the lower ends of the leaf stalks are swollen, so that the structure resembles a bulb. This is eaten raw or cooked, according to cultivar, and the finely divided leaves are sometimes used separately as a garnish. The flavour is similar to that of aniseed. Trimmed Florence fennel has storage requirements similar to those of celery.



222 Bacterial soft rot of fennel



223 Grey mould rot and watery soft rot of fennel

1 CAPPELLINI R.A., CEPONIS M.J. & LIGHTNER G.W. (1987) Disorders in celery and carrot shipments to the New York market, 1972–1985. *Plant Disease* **71,** 1054–1057.

2 COX R.S. (1958) Etiology and control of celery diseases in the Everglades. *Bulletin of Florida Agricultural Experiment Station* No. 598, 27 pp illus.

3 FOSTER A.C. & WEBER G.F. (1924) Celery diseases in Florida. *Bulletin of Florida Agricultural Experiment Station* No. 173, 77 pp.

4 GRIZZELL W.G. & HENRY F.E. (1971) A central packing-precooling system for celery. *Marketing Research Report of the United States Department of Agriculture* No. 869, 34 pp.

5 MIDDLETON J.T., KENDRICK J.B. & HALL B.J. (1952) Parasitic and nonparasitic diseases of celery in southern California. *Plant Disease Reporter* **36,** 188–193.

6 NOVIELLO C., MARZIANO F., ALOJ B. & GARIBALDI A. (1975) Osservazioni triennali sulle malattie del finocchio in Campania. *Annali della Facoltà di Scienze Agrarie della Università degli Studi di Napoli, Portici 9/10,* 259–271 illus.

7 PARSONS C.S. (1960) Effects of temperature, packaging and sprinkling on the quality of stored celery. *Proceedings of the American Society for Horticultural Science* **75,** 463–469.

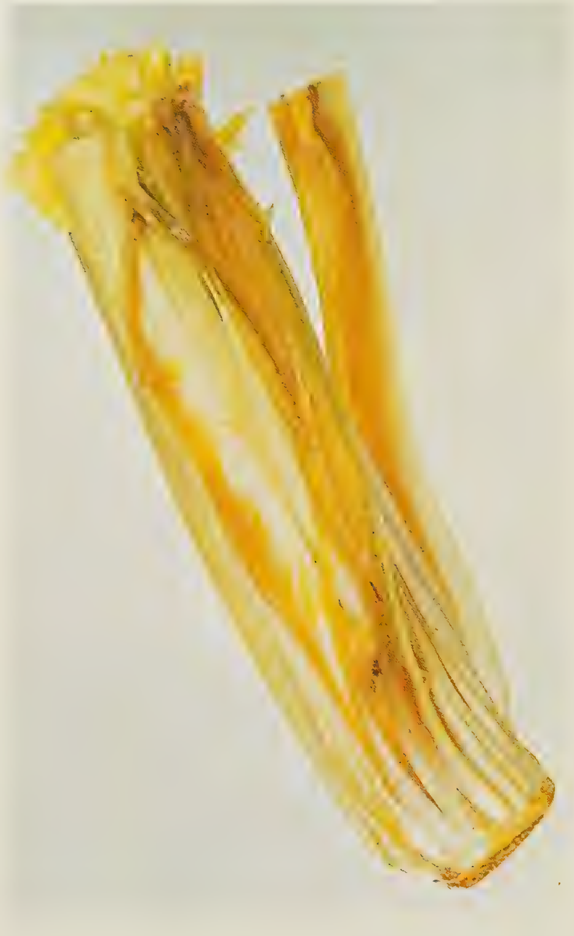
8 ROELOFSE E.W., HAND D.W. & HALL R.L. (1989) The effect of daylength on the development of glasshouse celery. *Journal of Horticultural Science* **64,** 283–292.

9 SMITH R.B. & REYES A.A. (1988) Controlled atmosphere storage of Ontario-grown celery. *Journal of the American Society for Horticultural Science* **113,** 390–394.

10 STEWART J.K. (1976) Transit temperatures and quality of fresh vegetables shipped in an experimental and a commercial van container to the Far East. *Marketing Research Report of the United States Department of Agriculture* No. 1054, 8 pp.

BACTERIAL SOFT ROTS of celery and fennel caused by
Erwinia sp.
Pseudomonas spp.

224



OCCURRENCE *Erwinia carotovora* ssp. *carotovora* (page 271) is widespread; for example, it has been recorded on celery in Canada (4) and Australia (5), and on fennel in Switzerland and Italy (3). *Pseudomonas fluorescens* and *P. marginalis* pv. *marginalis* (page 142) occur on celery in the UK and on fennel and celery in Italy (1,2).

SYMPTOMS Lesions may occur on any part of the plant and are at first watersoaked, later brown, moist, extremely soft and sometimes slimy.

BIOLOGY These bacteria are common on plant debris in the soil, and thrive in wet conditions (2). They are disseminated by water and by insects (4). Infection occurs via growth cracks (3), wounds caused by abrasion or insect activity (1,4) or through tissue weakened by physiological disorder such as blackheart (q.v.) (4). Injuries sustained at harvest-time are vulnerable to infection during post-harvest washing (5).

CONTROL It is important to have good field drainage, and excessive irrigation should be avoided, especially during hot weather (2) or late in the season (3). Appropriate application of fertilizer may be necessary to prevent physiological disorder, and insects should be controlled (4). Some fungicide sprays are effective against bacteria (5). If the harvested plants are to be washed, the water should be chlorinated and checked frequently for its bactericidal activity (since chlorine is rapidly inactivated in the presence of soil and plant debris). Rapid cooling of harvested produce is essential; refrigeration provides temporary control of *E. carotovora*, but severe symptoms develop on removal to higher temperatures (5). *P. fluorescens* and *P. marginalis* can make significant progress at 0° to 1°C (1) and consignments with incipient infection must therefore be marketed without delay.

---

1 BROCKLEHURST T.F. & LUND B.M. (1981) Properties of pseudomonads causing spoilage of vegetables stored at low temperature. *Journal of Applied Bacteriology* **50**, 259–266.

2 DEALTO A. & SURICO G. (1982) Effetto della temperatura e della umidità del terreno sulla sopravvivenza di due pseudomonadi pectolitiche fluorescenti isolate da piante e da rizosfera di sedano. *Phytopathologia Mediterranea* **21**(2/3), 50–58.

3 MAZZUCCHI U. & DALLI A. (1974) Bacterial soft rot of fennel (*Foeniculum vulgare* var. *dulce* Mill.). *Phytopathologia Mediterranea* **13**(1/2), 113–116.

4 RICHARDSON J.K. (1938) Studies on blackheart, soft-rot, and tarnished plant bug injury of celery. *Canadian Journal of Research C* **16**, 182–193 illus.

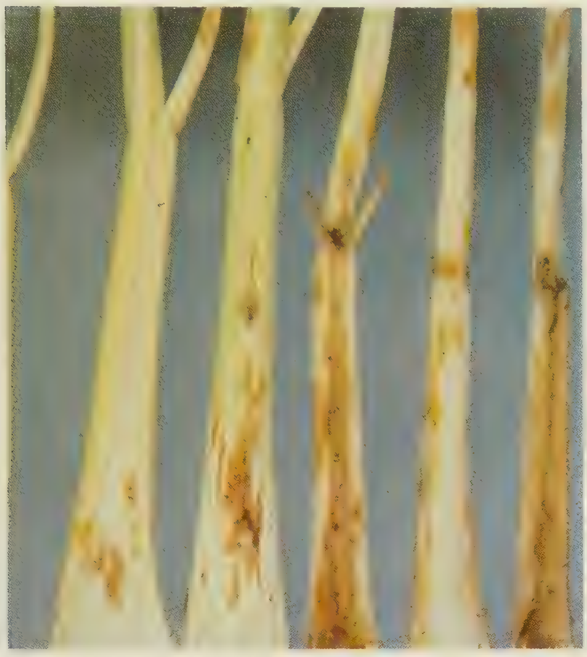
5 WIMALAJEEWA D.L.S. (1976) Studies on bacterial soft rot of celery in Victoria. *Australian Journal of Experimental Agriculture and Animal Husbandry* **16**, 915–920.

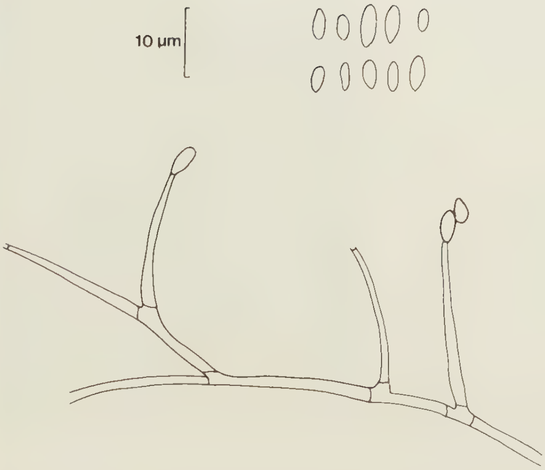
BROWN SPOT of celery caused by

***Acremonium apii* (M.A. Smith & Ramsey) Gams**

(syn. *Cephalosporium apii* M.A. Smith & Ramsey)

25





226 *Acremonium apii* (from CMI Descr. No. 932, Williams 1987) Conidiophore, conidiogenous cells, and conidia

OCCURRENCE Brown spot of celery has been recorded in several parts of the USA (1,2,3, 5), Canada and the UK (4).

SYMPTOMS The outermost stalks are especially vulnerable, lesions on the concave surface being rust-coloured, elongated, dry and slightly depressed, while on the convex side they tend to be more reddish-brown, smaller in size and fewer in number (4) or absent (3). Small transverse cracks may develop, in which there is copious production of spores (conidia) and, under humid conditions, there is a visible growth of white mould (5).

BIOLOGY The fungus can survive as resting spores (chlamydospores) in the soil or as mould strands in crop debris (1,4). During warm wet weather abundant asexual spores (conidia) are produced, dispersal being by water (5). Under moist conditions infection can occur over a wide range of temperature, but disease is severe only during seasons when the prevailing temperature is around 25°C (1,3). If infected celery is placed in store, fungal decay is frequently followed by bacterial soft rot (q.v.) (2).

CONTROL Fungicide sprays have been used successfully (4) but are not always effective (1). If an infected crop is to be stored or transported long distances, refrigeration serves to hold the disease in check. Brown spot does not appear to affect related crops such as fennel, carrots, parsnips or parsley (5), and since in the absence of celery crops the fungus persists in the field for only a few months, it is feasible to use crop rotation as a means of control (1). Some cultivars are resistant to the disease (1).

---

1 LEWIS G.D. (1959) Effects of temperature and crop rotation on the occurrence of brown spot of celery in southern New York. *Plant Disease Reporter* **43,** 1079–1080.

2 RICH S. (1958) Brown spot of celery in Connecticut. *Plant Disease Reporter* **42,** 1091–1092.

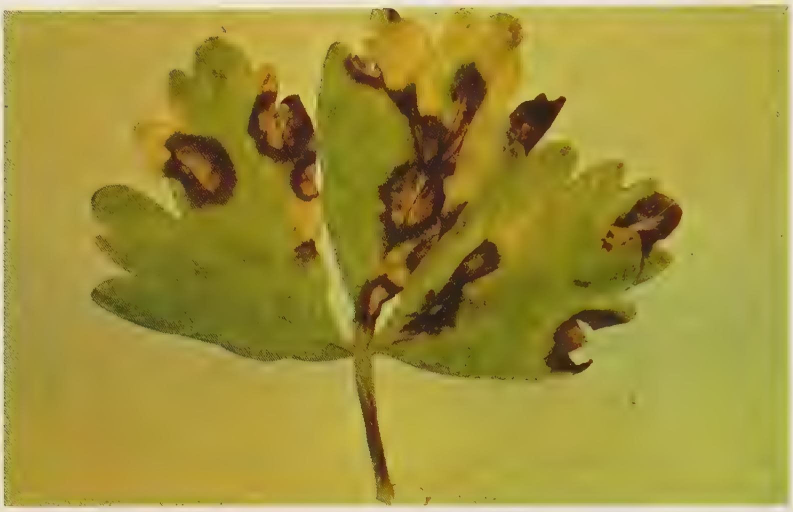
3 SEGALL R. (1951) Brown spot disease of celery found in New York. *Plant Disease Reporter* **35,** 164 illus.

4 SHERIDAN J.E. (1964) Brown spot of celery in England. *Plant Pathology* **13,** 161–163 illus.

5 SMITH M.A. & RAMSEY G.B. (1951) Brown-spot disease of celery. *Botanical Gazette* **112,** 393–400 illus.

CERCOSPORA SPOT or **EARLY BLIGHT** of celery caused by *Cercospora apii* Fres.

227



OCCURRENCE This disease may occur wherever celery is grown, for example many parts of the USA (2,3,4,5), South America (1), Europe and Australasia.

SYMPTOMS Spots on leaflets are circular and pale yellow, turning brown. Under humid conditions there is a fine growth of greyish mould at the centre of each spot. Lesions on stalks are similar except for their elongated shape. There are no black bodies such as are found in 'late blight' lesions (septoria spot, q.v.) (1).

BIOLOGY The fungus is sometimes seedborne. It also survives in crop debris, and its spores (conidia) are spread by watersplash, air currents, and the implements and clothing of farm labourers (2,3). Infection occurs via the natural openings (stomata) in leaves and stalks. Optimal conditions for disease development are a high relative humidity (2) and temperatures between 25° and 30°C (3), i.e. warmer conditions than those favouring late blight. Epidemics are likely when heavy evening dews follow hot days, especially if plants are suffering from lack of water (3).

CONTROL Clean or treated seed should be used, and it may be necessary to disinfect the seed-bed. Only healthy seedlings should be transplanted (2). Well-irrigated vigorous plants can better withstand attack by the early blight fungus (3). Spacing out the plants or growing them on raised beds (to reduce humidity through improved air circulation) does not appear to be commercially feasible (4). Frequent spraying with fungicide is the chief method of controlling this disease (1,4). Monitoring numbers of spores in the atmosphere helps the forecasting of potential outbreaks and hence the correct timing of sprays (2). Some celery cultivars are partially resistant to early blight (1).

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1 ALIPPI H.E. (1965) Tizón temprano o cercosporiosis del Apio (*Cercospora apii* Fres.). *Revista de la Facultad de Agronomía de la Universidad Nacional de la Plata* **41**, 51–56 illus.

2 BERGER R.D. (1973) Early blight of celery: analysis of disease spread in Florida. *Phytopathology* **63**, 1161–1165.

3 KLOTZ L.J. (1923) A study of the early blight fungus *Cercospora apii* Fres. *Technical Bulletin of Michigan Agricultural Experiment Station* No. 63, 43 pp illus.

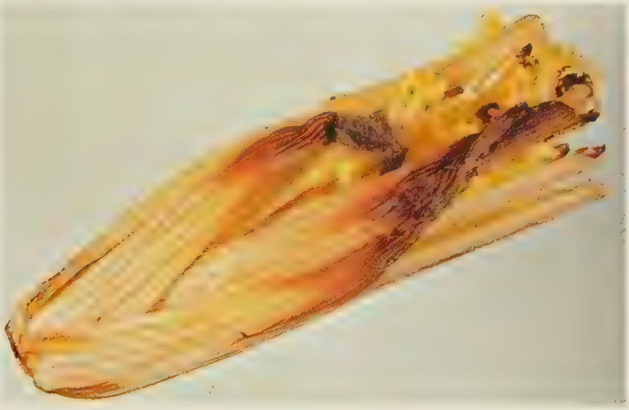
4 STRANDBERG J.O. & WHITE J.M. (1978) *Cercospora apii* damage of celery – effect of plant spacing and growth on raised beds. *Phytopathology* **68**, 223–226.

5 WOLF E.A. (1970) Earlibelle, a new early blight resistant celery. June-Belle, a new early blight resistant celery for spring harvest in south Florida. *Circulars of Florida Agricultural Experiment Station* Nos. S207, 9 pp; S208, 9 pp.

GREY MOULD ROT of celery and fennel caused by

***Botryotinia fuckeliana* (de Bary) Whetzel**

Conidial state: *Botrytis cinerea* Pers.



228

OCCURRENCE Grey mould rot probably occurs in all countries where celery and fennel are stored for more than a few weeks. The disease has been reported on celery in the USA, the UK (2,3), Romania (4) and Israel (1). The author has observed it on fennel imported from Italy and held in store in the UK.

SYMPTOMS Lesions are soft and watery, reddish-brown or buff, and in the early stages may resemble those of watery soft rot or pink rot (q.v.). Later, however, grey mould rot is easily distinguished by the copious production of grey-brown spores (conidia) and the formation of small black resting bodies (sclerotia) which are characteristically embedded in the affected tissue.

BIOLOGY The fungus survives in the soil as sclerotia, and also as mould strands in plant debris left in the field. During mild damp weather numerous spores are produced, and harvested plants are likely to be contaminated when placed in store, especially if much soil and leaf debris is included (2). Freshly trimmed plants are resistant to attack, but as the tissue ages it becomes susceptible to direct invasion by the fungus (1,3); thus decay usually starts on the outermost stalks of celery (especially at injuries) and the fading leaves of fennel, later spreading to healthy plants in contact. The optimal growth temperature for the fungus is approximately 23°C, but over a period of weeks it can make substantial progress even at 2°C (2).

CONTROL Plants should be properly spaced, since a dense mass of foliage harbours moisture which encourages spore formation on moribund tissue (4). It is advantageous if the plants can be harvested under conditions which permit easy removal of soil (2). Careful trimming should precede long-term storage, and post-harvest treatments are beneficial; a disinfectant should be added to washing water, and the produce can be dipped in a growth-regulating chemical to delay senescence of the tissue, together with a fungicide to retard rotting (1). Prompt cooling is important; high humidity and a temperature close to 0°C serve to minimise deterioration in store. Controlled atmosphere storage has potential, and removal of ethylene is especially beneficial (5).

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1 BARKAI-GOLAN R. & AHARONI N. (1981) GA_3 for the control of soft rots on stored celery. *Proceedings of the 5th Congress of the Mediterranean Phytopathological Union, Patras, Greece 1980*, pp. 167–169. Athens: Hellenic Phytopathological Society.

2 DERBYSHIRE D.M. & CRISP A.F. (1971) Vegetable storage diseases in East Anglia. *Proceedings of the 6th Insecticide and Fungicide Conference*, pp. 167–172.

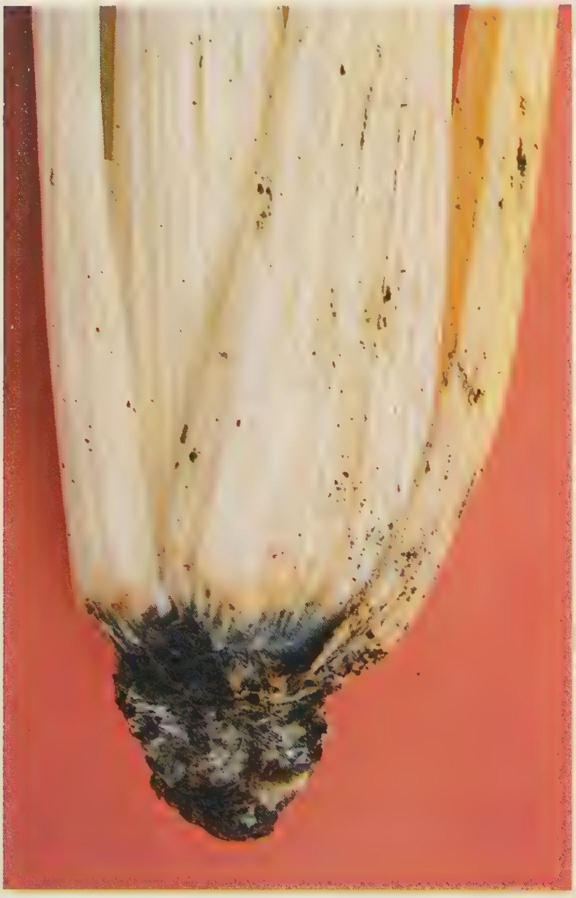
3 LOWINGS P.H. (1955) Some storage rots of celery caused by *Centrospora acerina* and other fungi. *Plant Pathology* **4**, 106–107.

4 RAICU C. & STAN G. (1976) [Diseases of celery grown for stalk and leaves in greenhouses.] *Producţia Vegetală, Horticultura* **25**(3), 35–37 illus.

5 REYES A.A. & SMITH R.B. (1986) Controlled atmosphere effects on the pathogenicity of fungi on celery and on the growth of *Botrytis cinerea*. *HortScience* **21**, 1167–1169.

LICORICE ROT of celery caused by
Mycocentrospora acerina **(Hartig) Deighton**

229



OCCURRENCE Licorice rot, which characteristically appears late in storage, is a serious disease of celery in the UK (1,3), the USA (4) and Canada (5). It also occurs on celeriac in Germany (2).

SYMPTOMS Elongated lesions are occasionally found on the upper parts of the stalks, but more usually decay begins near the butt (1,3). Infected tissue is initially brown but later darkens to greenish-black, and the symptoms resemble those of phoma rot (q.v.) (4). Microscopic examination reveals an abundance of dark spores (chlamydospores) and, occasionally, asexual spores (conidia) which characterise the licorice rot fungus (page 285) (2,4). As decay advances along the stalks, the rotten butt may fall away completely (3,5).

BIOLOGY The fungus survives in the soil as chlamydospores, and celery harvested from infested fields is likely to be contaminated when placed in store. Freshly trimmed celery is highly resistant to infection by this fungus, but after several weeks becomes susceptible to invasion, either through existing wounds or by direct penetration of ageing tissue (1,5). Infection and decay take place even at 0°C (2), and severe losses can occur after long-term cold storage (3). The evidence is, however, that the disease does not spread from plant to plant in store (1,5).

CONTROL Because of the lag phase in infection, no control measures are necessary if the celery is to be marketed immediately or cold-stored for only five or six weeks (1). If long-term storage is envisaged, then a post-harvest fungicide dip is essential, since refrigeration alone does not provide adequate control.

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1 DAY J.R., LEWIS B.G. & MARTIN S. (1972) Infection of stored celery plants by *Centrospora acerina*. *Annals of Applied Biology* **71**, 201–210 illus.

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4 NEWHALL A.G. (1944) A serious storage rot of celery caused by the fungus *Ansatospora macrospora* n. gen. *Phytopathology* **34**, 92–105 illus.

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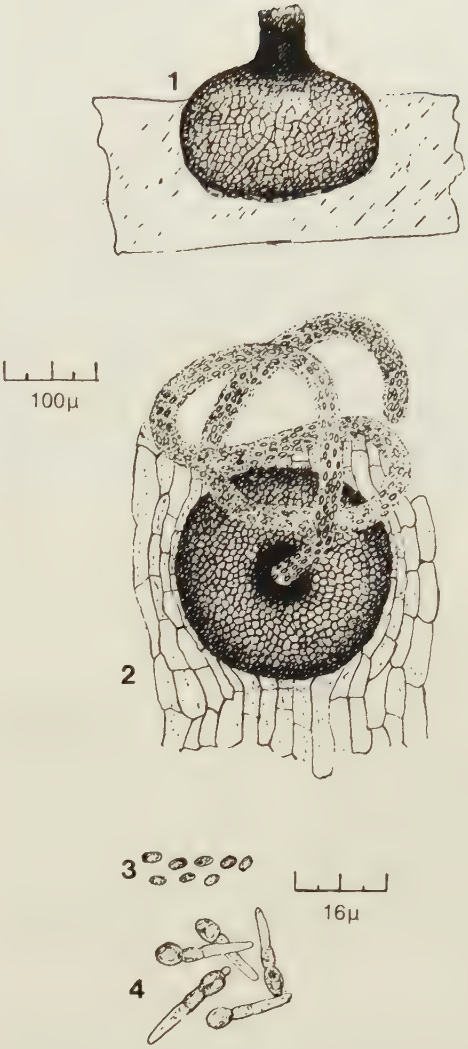
PHOMA ROT of celery and fennel caused by *Phoma apiicola* Klebahn

OCCURRENCE Phoma rot of celery has been reported from various parts of the USA (1,2), the UK (4), Germany (3), France and the Netherlands (2). The fungus is also capable of attacking celeriac (5) and fennel (3).

SYMPTOMS The butt becomes soft, brown (1) or blueish-green (2) and later black as decay spreads into the stalks. At this stage the symptoms resemble those of licorice rot (q.v.); however, in advanced phoma rot, minute spherical black bodies (pycnidia) are produced and eventually erupt through the skin (2,4).

BIOLOGY The fungus survives on crop debris in the asexual state (pycnidia giving rise to conidia), and spores are disseminated in rain or irrigation water. Infection of the butt takes place during cool wet weather (1), the optimal growth temperature for the fungus being approximately 18°C (2). Although young plants can be attacked directly, infection of mature plants usually takes place via wounds (2) such as those made by insects. Decay continues after harvest.

CONTROL Although there are occasional reports of seed transmission, most outbreaks originate from infected soil (2,4). It was earlier reported that, since infection occurred below the soil surface, spraying the plants was useless (2). However, modern systemic fungicides show increased effectiveness. Alternatively it may be feasible to sterilize the soil (5). Traditional means of control have been mainly cultural, such as prompt collection and destruction of plant refuse, coupled with crop rotation, excluding other susceptible crops such as carrots, parsnips and parsley (2,5).



230 *Phoma apiicola* (from *Tech. Bull. Mich. agric. Exp. Stn* No. 53, p. 33, Bennett 1921) 1 pycnidium 2 spores (conidia) exuding from pycnidium 3 conidia 4 germinating conidia

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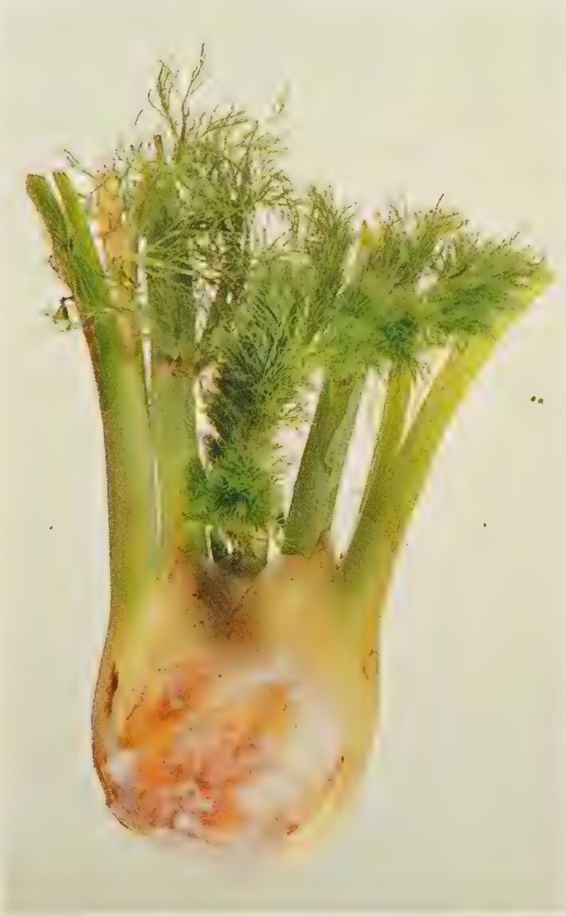
1 BARDIN R. & FONG R. (1947) Phoma root and crown rot of celery. *Bulletin of the State of California Department of Agriculture* No. 36, 105–106 illus.
2 BENNETT C.W. (1921) A Phoma root rot of celery. *Technical Bulletin of Michigan Agricultural Experiment Station* No. 53, 40 pp illus.
3 GERSTER W. (1952) Untersuchungen über die Biologie des Erregers des Sellerieschorfes (*Phoma apiicola* Klebahn). Wissenschaftliche Zeitschrift der Martin-Luther-Universität, Halle-Wittenberg 2, 83–126.
4 HEWETT P.D. (1971) *Phoma apiicola* on celery seed. *Plant Pathology* **20,** 123–126.
5 WILLIAMS P.H. & WADE E.K. (1967) Scab of celeriac. *Plant Disease Reporter* **51,** 427–429.

PINK ROT or **WATERY SOFT ROT** of celery and fennel caused by

Sclerotinia minor **Jagger**
Sclerotinia sclerotiorum **(Lib.) de Bary**

231



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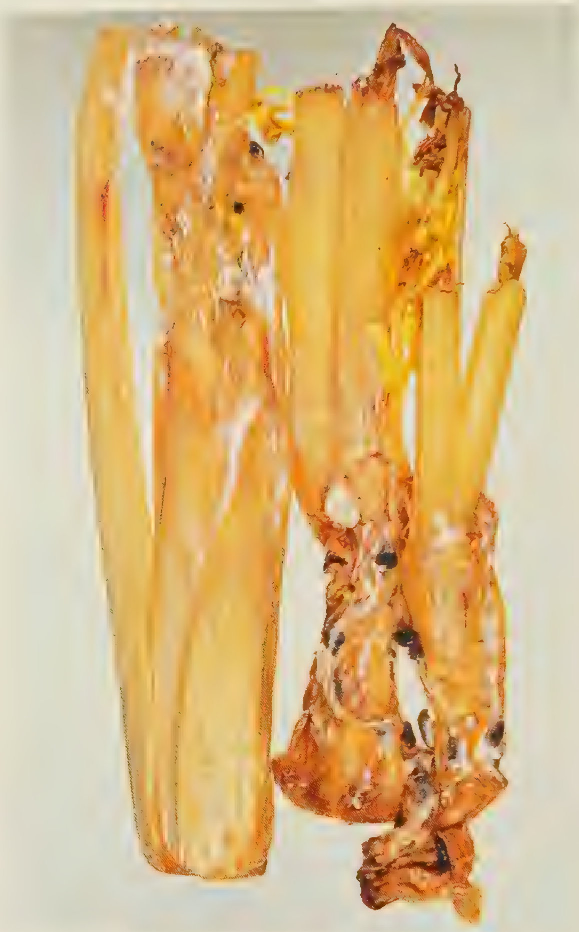

OCCURRENCE *Sclerotinia sclerotiorum* causes severe losses of celery in Canada (10), the USA (3,9), Argentina (4) and Israel (2), and has also been reported in the UK (5) and other parts of Europe. Fennel is affected in Italy (1) and also, during the cool season, in Egypt and India. *S. minor* has been recorded on celery in the USA (6) and on fennel in Italy (7).

SYMPTOMS Lesions are pale brown with a pinkish-brown border, and a watery liquid may leak from infected tissues; handling of infected plants should be avoided, in view of well-documented cases of skin-rashes suffered by people harvesting infected celery (8). Severely rotted plants become covered with a bright white mould, on which are formed resting bodies (sclerotia), at first white and later black. Those of *S. sclerotiorum* have dimensions of several mm while those of *S. minor* are from 1 to 2 mm (6,7).

BIOLOGY These fungi can persist in the soil for many years as sclerotia (9). A few weeks after the onset of cool wet weather, those of *S. sclerotiorum* (page 47) (4), and occasionally those of *S. minor* (6), 'germinate' to produce the sexual stage, in the form of small cup-shaped structures (apothecia giving rise to ascospores) (3,5,9). Spores are ejected into the air and lodge in the foliage or are washed down by rain or irrigation water towards the base of the plant, where they develop into strands of mould. Alternatively, and more usually in *S. minor*, mould strands grow out directly from sclerotia (6). If wet conditions continue then infection takes place, generally at soil-level, and there may be severe losses in the field (4,9). Plants with incipient infection undergo rotting in store, since most strains of these fungi are capable of growth even at temperatures close to 0°C (10). Decay subsequently spreads to healthy plants in contact, and leaking juices cause further spoilage.

233

CONTROL Deep ploughing serves to bury sclerotia, but is not always effective since they can remain viable for many years (3,9). Flooding the land for several weeks is a surer method, since most of the sclerotia are thereby killed (3). In countries with a hot dry season it may be feasible to use the technique known as 'solarization' (page 132); the fields are covered with polyethylene for several weeks, between crops, and soil temperatures rise to levels which are lethal to sclerotia. Alternatively, fungicides may be applied to the soil or to the growing crop (1,3,5,9). A simple cultural method in dry growing areas involves 'earthing up' the plants and watering them from below by channel or trickle irrigation; the top soil round the plants is thereby kept dry and infection is discouraged. Crops from infested fields may be dipped in a fungicide (2), and prompt cooling helps to minimise rotting in store. Controlled atmosphere storage may be used as a supplement to refrigeration (10).

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1 ALOJ B. & GARIBALDI A. (1978) Esperienze di lotta in Campania contro la *Sclerotinia sclerotiorum* (Lib.) de Bary, agente di un marciume del grumolo del finocchio. *Annali della Facoltà di Scienze Agrarie della Università degli Studi di Napoli, Portici* **12,** 213–216.

2 BARKAI-GOLAN R. & AHARONI N. (1972) Postharvest thiabendazole treatments to prevent spoilage in stored celery. *Israel Journal of Agricultural Research* **22,** 157–159.

3 BROOKS A.N. (1940) Control of sclerotiniose of celery on Florida muck. *Phytopathology* **30,** 703 [Abstract].

4 FELDMAN J.M. & PONTIS R.E. (1960) La podredumbre rosada del apio en la Argentina causada por *Sclerotinia sclerotiorum. Revista Argentina de Agronomía* **27,** 15–26 illus.

5 GRIFFIN M.J. (1985) Evaluation of dicarboximide fungicides for control of pink rot (*Sclerotinia sclerotiorum*) of glasshouse celery. *Tests of Agrochemicals and Cultivars* No. 6 (*Annals of Applied Biology* **106,** Supplement), 48–49.

6 JAGGER I.C. (1920) *Sclerotinia minor,* n.sp., the cause of a decay of lettuce, celery, and other crops. *Journal of Agricultural Research* **20,** 331–333 illus.

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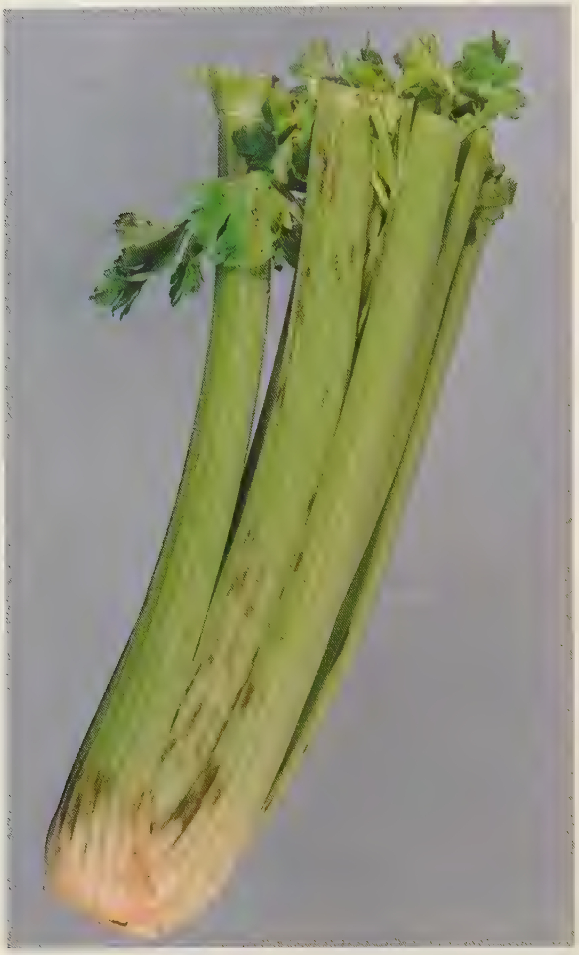
8 PERONE V.B., SCHEEL L.D. & MEITNS R.J. (1964) A bio assay for the quantitation of cutaneous reactions associated with pink-rot of celery. *Journal of Investigative Dermatology* **42,** 267–271.

9 POTTER H.S., CLONINGER C.K. & DROST A. (1958) Foliar and soil applications of chemicals for the control of pink rot of celery. *Quarterly Bulletin of Michigan Agricultural Experiment Station* **40,** 734–739 illus.

10 REYES A.A. (1988) Suppression of *Sclerotinia sclerotiorum* and watery soft rot of celery by controlled atmosphere storage. *Plant Disease* **72,** 790–792.

SEPTORIA SPOT or **LATE BLIGHT** of celery caused by *Septoria apiicola* Speg.

234



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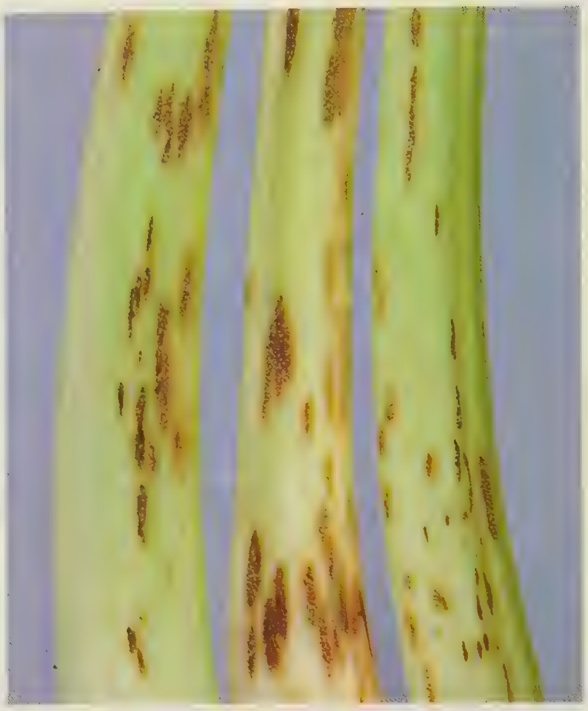
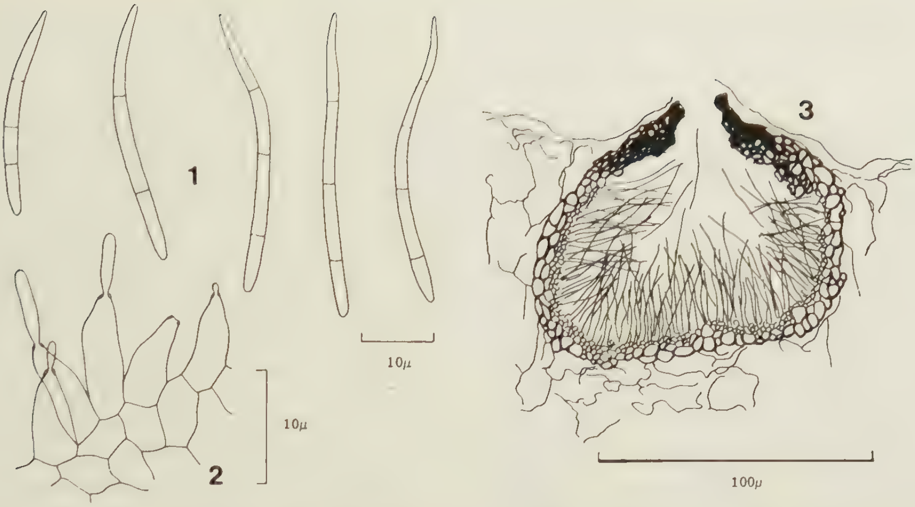


OCCURRENCE This disease may occur wherever celery is grown (4), for example the USA (2,8), the UK (5,9), Italy (1), Poland (6), Belgium (10) and Australia (3,7).

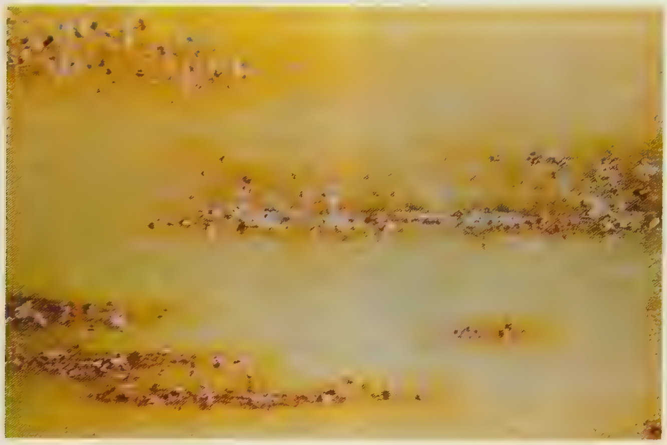
SYMPTOMS Spots on leaflets are circular, yellow at first and later brown or grey, while on stalks the lesions are elongated and brownish, without a definite margin (4). Minute black bodies (pycnidia) develop in the affected areas (3).

BIOLOGY The fungus exists in the asexual state (pycnidia giving rise to conidia). Celery seed may be infected or contaminated (2,5) and, in some growing areas, the disease may first be manifest at the seedling stage. The fungus also persists in debris from previous infected celery crops (9). Spores are disseminated by rainsplash or irrigation water (2), and infection occurs via natural openings (stomata) or by direct penetration of leaf and stalk tissue (6). The resulting lesions give rise to more spores, which ooze out during humid weather. Secondary spread can occur when labourers move through the crop, and infection can be severe after a heavy dew. Moist conditions and temperatures around 20°C are optimal for disease development (6). In the northern USA such weather is most likely at the end of the growing season, hence the common name of 'late blight' (4). In other areas, however, such conditions may occur at the beginning of the season, so that 'late blight' may appear before 'early blight' (cercospora spot, q.v.).

CONTROL Crop rotation is beneficial, celeriac and wild celery being the only other hosts of this fungus (9). It is also good practice to remove plant debris at the end of the season. Seed harvested from an infected crop should be steam-treated (7) or soaked in a bath of fungicide (5). These measures may not be sufficient, and it is generally necessary to spray the growing crop with fungicides (1,3,8,10). Excessive application of nitrogenous fertilizer should be avoided. Development of incipient infections in harvested celery may be inhibited by rapid cooling and maintenance of low temperature during transport and storage. Some celery cultivars are resistant to late blight.



236 *Septoria apiicola* (from CMI Descr. No. 88, Sutton & Waterston 1966) 1 conidia 2 conidiophores 3 pycnidium



237 Spore tendrils (conidia exuding from pycnidia in celery tissue)

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1 ALOJ B. & GARIBALDI A. (1982) Prove di lotta contro la septoriosi del sedano in Campania. *Annali della Facoltà di Scienze Agrarie della Università degli Studi di Napoli, Portici* **16,** 34–38.

2 BERGER R.D. (1970) Epiphytology of celery late blight. *Proceedings of Florida State Horticultural Society* **83,** 208–212.

3 DULLAHIDE S.R. (1979) *Septoria* leaf spot control in celery. *Queensland Agricultural Journal* **105,** 317–320 illus.

4 GABRIELSON R.L. & GROGAN R.G. (1964) The celery late blight organism *Septoria apiicola. Phytopathology* **54,** 1251–1257 illus.

5 GOTT K.A., MAUDE R.B. & THOMAS T.H. (1989) Fungitoxicity of plant growth regulators (PGRs) and PGR/fungicide mixtures in soak treatments to *Septoria apiicola* pycnidiospores. *Plant Pathology* **38,** 21–25.

6 KOCHMAN J. & KUBICKA H. (1961) [Some biological study on *Septoria apiigraveolentis* Dorogin.] *Acta Agrobotanica* **10**(1), 99–122 illus.

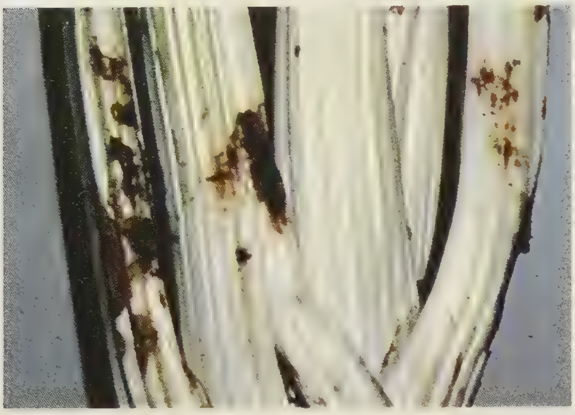
7 NAVARATNAM S.J., SHUTTLEWORTH D. & WALLACE D. (1980) The effect of aerated steam on six seed-borne pathogens. *Australian Journal of Experimental Agriculture and Animal Husbandry* **20,** 97–101.

8 PAULUS A.O., NELSON J. & OTTO H. (1979) Combination versus single fungicides for control of *Septoria* leafspot in celery. *California Agriculture* **33**(6), 15.

9 SHERIDAN J.E. (1966) Celery leaf spot: sources of inoculum. *Annals of Applied Biology* **57,** 75–81.

10 VULSTEKE G. & MEEUS P. (1986) New fungicides for the control of *Septoria apiicola* in celery. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **93,** 237–245.

OTHER DISEASES of celery and fennel



238 Crater rot of celery

BACTERIAL BLIGHT of celery may be caused by *Pseudomonas syringae* pv. *apii* (Jagger) Young *et al.* or by *P. cichorii* (Swingle) Stapp. Optimal temperatures for infection are 20°C and 30°C respectively (9), and prior wounding is necessary (4). Leaf spotting is common (4) and there may also be elongated rusty brown lesions (1 to 2 mm by 2 to 5 mm) on the stalks (10). Control is by seed-treatment and spraying.

BLACK ROT of celery, caused by *Alternaria radicina* (page 273), is usually a minor disease, but on some small-holdings with limited rotation it can become persistent and damaging (2). Infected plants may be partially or completely girdled. Control involves a careful choice of fungicide, since some chemicals can aggravate disease severity (2).

CRATER ROT of celery is caused by *Thanatephorus cucumeris* (sclerotial state: *Rhizoctonia solani*) (page 307), and has been recorded in the UK and the USA (3,7). Uninjured tissue below ground level may be directly invaded by the fungus, which causes oval brown sunken lesions on the lower parts of the stalks (3). Both the sexual state (basidiospores) and the sclerotial state have been observed. Often there is secondary decay by *Botrytis cinerea* (grey mould, q.v.) or *Erwinia carotovora* (causing bacterial soft rot, q.v.) (3). Control entails efficient field drainage and timely application of fungicide sprays (7).

FUSARIUM ROT of celery is caused by *Fusarium oxysporum* f.sp. *apii* (Nelson & Sherb.) Snyder & Hansen, which also causes 'yellows' in the growing crop. Infection occurs via the roots, and the water-conducting tissues of the plant are discoloured; furthermore the butt may undergo a brown to black dry rot (6). Control has been achieved by steam-sterilization of the soil. Certain rotations (e.g. with onions) are reported to be beneficial (1).

PHYTOPHTHORA ROT of fennel is caused by *Phytophthora syringae* (Kleb.) Kleb. and has been recorded in Italy. Under wet conditions infection of foliage occurs and decay may spread into the basal part of the plant (5). The optimal growth temperature for the fungus is approximately 20°C but it can develop slowly even at temperatures close to 0°C. The disease is controlled by spraying the crop with a fungicide.

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1 ELMER W.H. & LACY M.L. (1987) Effects of crop residues and colonization of plant tissues on propagule survival and soil populations of *Fusarium oxysporum* f.sp. *apii* Race 2. *Phytopathology* **77,** 381–387.

2 ESPIR A.F., GRIFFIN M.J., JONES D.R. & LOCKLEY K.D. (1985) Evaluation of fungicides for control of black rot (*Alternaria radicina*) of outdoor celery. *Tests of Agrochemicals and Cultivars* No. 6 (*Annals of Applied Biology* **106,** Supplement), 50–51.

3 HOUSTON B.R. & KENDRICK J.B. (1949) A crater spot of celery petioles caused by *Rhizoctonia solani. Phytopathology* **39,** 470–474 illus.

4 JAGGER I.C. (1921) Bacterial leafspot disease of celery. *Journal of Agricultural Research* **21,** 185–188 illus.

5 NOVIELLO C. & SNYDER W.C. (1962) A Phytophthora disease of fennel. *Phytopathologische Zeitschrift* **46,** 139–163 illus.

6 OTTO H.W., PAULUS A.O., SNYDER M.J., ENDO R.M., HART L.P. & NELSON J. (1976) A crown rot of celery. *California Agriculture* **30**(6), 10–11 illus.

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9 THAYER P.L. (1965) Temperature effect on growth and pathogenicity to celery of *Pseudomonas apii* and *P. cichorii. Phytopathology* **55,** 1365.

10 WILKIE J.P. & DYE D.W. (1974) *Pseudomonas cichorii* causing tomato and celery diseases in New Zealand. *New Zealand Journal of Agricultural Research* **17,** 123–130 illus.

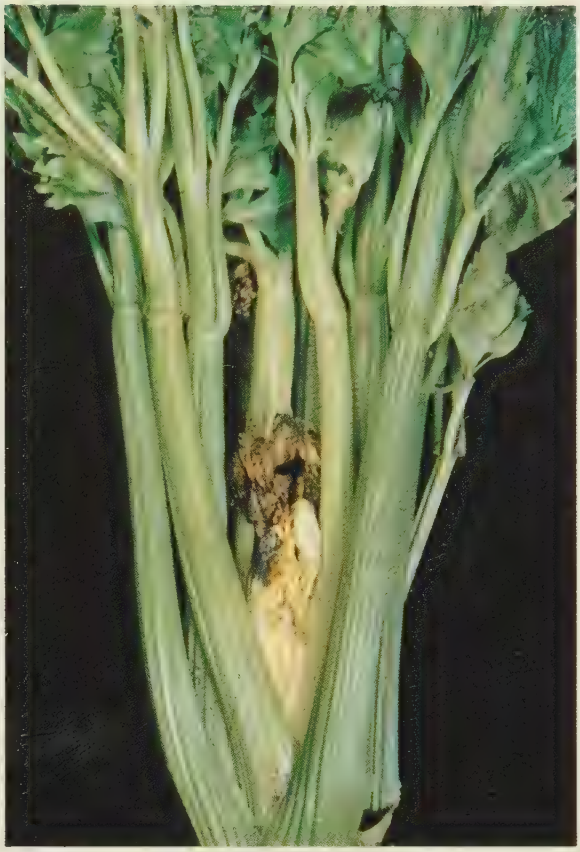
DISORDERS of celery

BLACKHEART occurs in most celery-producing areas and can cause heavy losses. Margins and tips of the heart leaves turn brown and later black. Symptoms appear during the latter part of the growing season, and may worsen during storage; not only is affected tissue unsightly, but secondary bacterial soft rotting may ensue. Blackheart of celery has much in common with tip-burn of lettuce (q.v.), being essentially a calcium deficiency disorder, although the relationship is far from simple (1). In water-stressed plants especially, the amount of calcium translocated to the rapidly expanding heart leaves may be insufficient. Celery cultivars differ in susceptibility to blackheart, and there are many other influential factors, such as other minerals supplied to the crop (1), and the temperature and humidity at harvest. Control measures include the application of a calcium spray or drench, and the careful management of fertilization and irrigation.

BLACK STEM is a post-harvest disorder of uncertain etiology, characterised by an unsightly blackening which appears as a striped pattern in the intact petiole and as black 'dots' in transverse section (4). It is the vascular strands which are affected. Controlled atmosphere storage can provide almost complete control (4).

BROWN CHECKING and **CRACKED STEM** result from boron deficiency or, more accurately, an imbalance in the ratio of boron and other elements (2,3). Cracks develop at right angles to the prominent ribs of the petioles, and affected tissue is discoloured brown. Control measures include the use of resistant cultivars, careful choice of fertilizers, and the judicious application of boron compounds.

FREEZING INJURY takes various forms (5). Severely frozen tissues appear wilted and water-soaked on thawing. Milder freezing causes depressions in the tissue which subsequently tend to turn brown. Celery freezes at approximately −0.5°C, petioles freezing more readily than leaves. Since the optimal storage temperature for celery is 0° to 1°C, it is essential to have accurate thermostats in refrigerated stores and vehicles if inadvertent freezing is to be prevented.



239 Blackheart of celery

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1 ALONI B. & PRESSMAN E. (1987) The effects of salinity and gibberellic acid on blackheart disorder in celery (*Apium graveolens* L.). *Journal of Horticultural Science* **62,** 205–209.

2 ATKINSON J.D. (1948) Cracked-stem of celery (*Apium graveolens* L.). *New Zealand Journal of Science and Technology A* **24,** 261–264 illus.

3 BURDINE H.W. (1973) The development of pencil stripe in celery. 2. Internal induction. A manifestation of brown checking. *Proceedings of the Soil and Crop Science Society of Florida* **32,** 77–83 illus.

4 WALSH J.R., LOUGHEED E.C., VALK M. & KNIBBE E. (1985) A disorder of stored celery. *Canadian Journal of Plant Science* **65,** 465–469 illus.

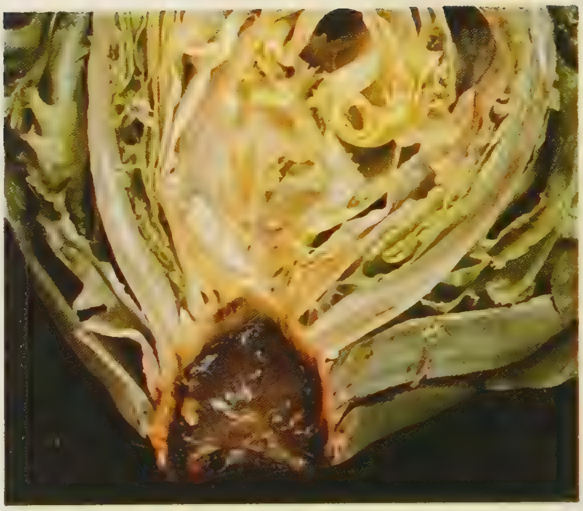
5 WHITE-STEVENS R.H. (1937) Some cellular changes in celery during freezing and frost hardening. *Proceedings of the American Society for Horticultural Science* **34,** 570–576.

Lettuce, chicory and endive

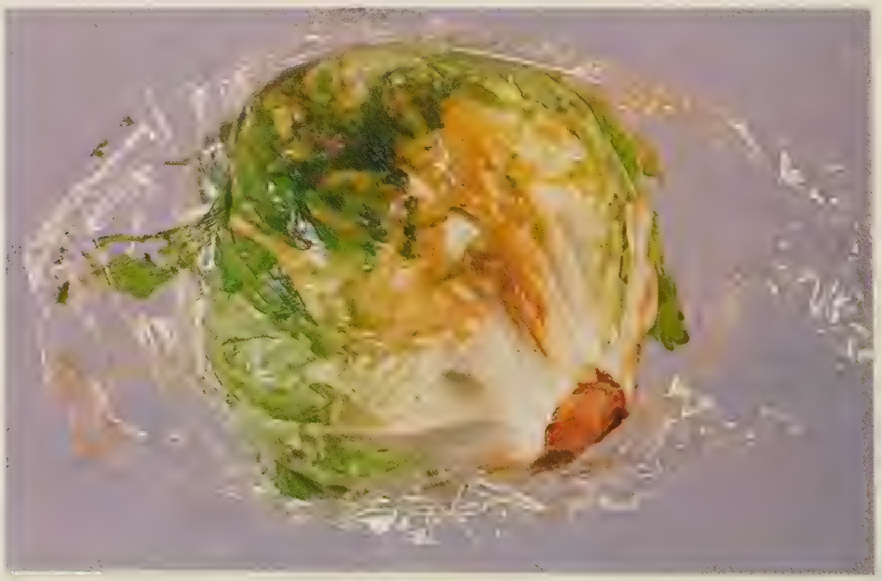
Lettuce, *Lactuca sativa* L., is a member of the family Compositae, and is native to the Mediterranean region, where it has been cultivated for several millennia. It probably spread into the rest of Europe during the Roman occupation. Once introduced into the Americas in the 15th century, lettuce became popular in the New World and is now the major salad crop in the USA, Canada and much of South America. It is important in most European countries, Australia and Japan.

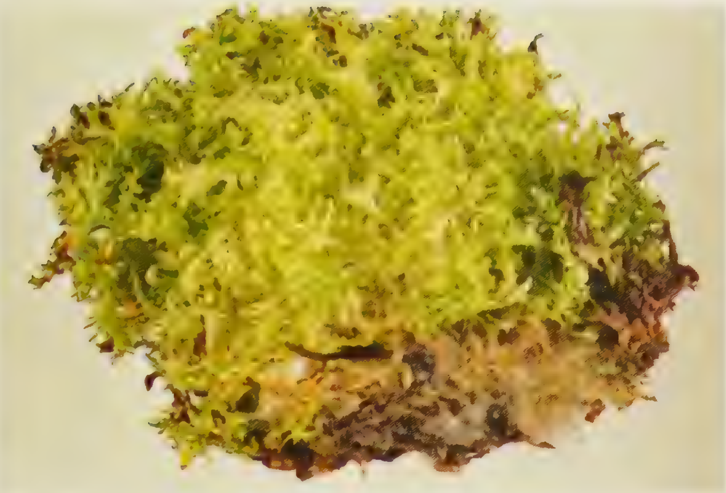
There are several types of lettuce, differing in leaf shape and form, and in the tendency to form a 'heart' or 'head'. Head or cabbage lettuce is *L. sativa* var. *capitata*. Butterheads are soft-leaved, while crispheads are firm with thick crunchy leaves. Crisphead (Iceberg) types are predominant in the USA, and are beginning to displace some of the traditional butterheads of northern Europe. Cos or romaine lettuce, *L. sativa* var. *longifolia*, is characterised by elongated upright heads with long stiff leaves possessing prominent mid-ribs. Leaf lettuce is *L. sativa* var. *crispa*, comprising non-heading types with curly, sometimes reddish, leaves. Stem lettuce or celtuce, *L. sativa* var. *angustana*, is a non-heading variety which is grown for its substantial fleshy stalk.

Lettuce is unique among the major vegetables in that it is used almost exclusively as a fresh raw product. It can be grown as a field crop or else under the protection of glass or plastic. Even in areas with adequate total rainfall, irrigation is necessary for the prevention of physiological disorders in mature heads (3, 7). Although sprinkling is appropriate for seedlings, surface irrigation is preferable for the developing crop, this method being less conducive to the spread of disease (7). Aphid infestation (1) can be a problem, and there is interest in mechanical suction devices as an alternative to the use of pesticides. In the greenhouse environment, biological control may be feasible.


240 Butt rot (bacterial) of lettuce

Harvesting is usually done by hand, though machines are available, both for 'once-over' and for selective harvesting. It is common practice for the packing operation to take place in the field. Trimmed heads may be packed 'naked' in cartons, or be individually film-wrapped by personnel working on a machine following the pickers. Demand for 'convenience foods' has led to a third alternative, in which the harvested heads are conveyed in bulk to a central station, where they are trimmed and shredded


241 Bacterial soft rot of lettuce

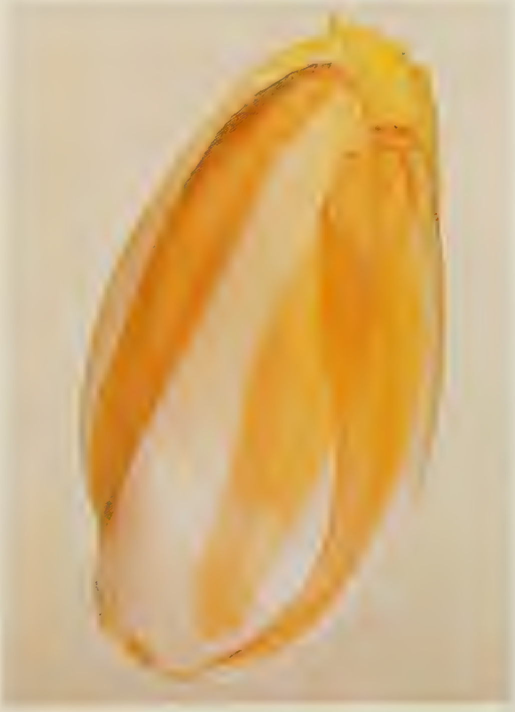


242 Grey mould rot of curly endive

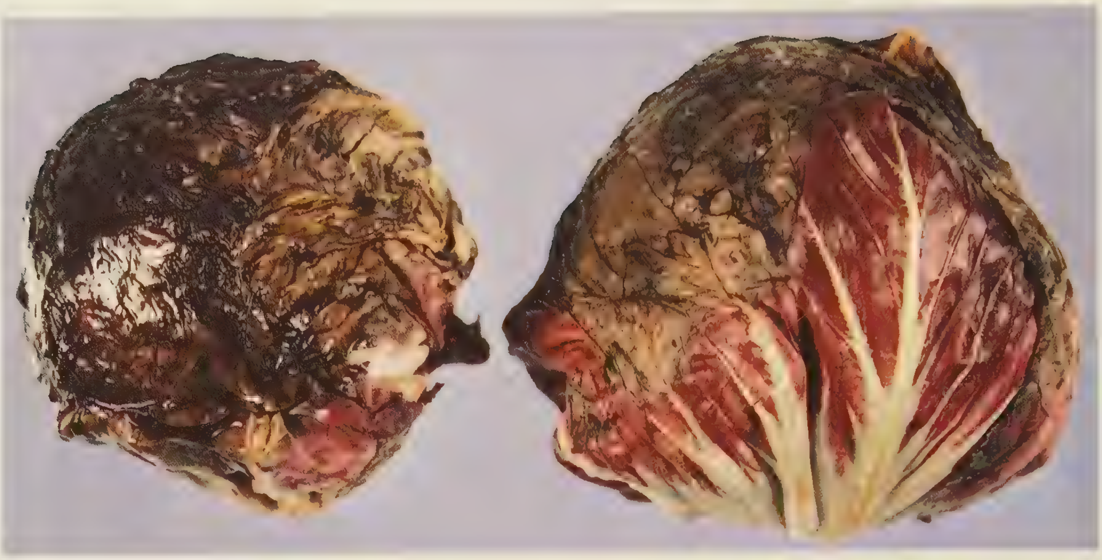
prior to cooling and packing. Whichever handling system is used, the aim should be to cool the lettuce as promptly as possible. The rate of respiration (and hence heat production) of leaf lettuce is approximately twice that of head lettuce. In view of the substantial surface area of the leaves, vacuum-cooling is especially appropriate; prior misting of naked lettuces with a water-spray ensures that moisture loss from the leaves is minimised. Hydro-cooling is also feasible, as is forced-air cooling if there is provision for maintaining atmospheric humidity close to saturation. Where aphid infestation is a problem, 100% mortality can be achieved by subjecting the load to reduced atmospheric pressure for 2 days (1).

The optimal storage temperature for lettuce is 0° to 1°C (7), and storage life decreases markedly at higher temperatures. On the other hand, care must be taken to avoid freezing injury, the critical temperature for lettuce being only a fraction of a degree below 0°C. It is for this reason that, as a precaution, carriage temperatures of 2°, 3° or even 4°C are sometimes requested for refrigerated trailer or container shipments of lettuce. It is preferable, however, to use vehicles and containers in which accurate temperature control can be guaranteed, and in which the thermostat is located in the air delivery rather than in the air return stream; by this means, freezing temperatures may be avoided (3). 'Bottom air delivery', in which cooled air is directed beneath the load, is a more efficient system than 'top air delivery', and helps to ensure thorough distribution of cooled air; otherwise there is a risk of 'hot spots' developing within the stow, especially during summertime shipment (3). Of all the commodities arriving in the New York market, lettuce was found to have the highest number of consignments rejected or discounted because of rots, physiological disorders, and injuries (3).

The use of controlled atmosphere storage, or modified atmosphere packaging, can extend the storage life of lettuce by several weeks (2,10). Cultivars differ greatly, however, in their response to increased carbon dioxide levels and reduced oxygen. Respiratory disorders include 'brown stain' and 'heart-leaf injury' (q.v.). 'Russet-spotting' (2,3,7) is held in check by low oxygen levels, and greatly aggravated by exposure to ethylene, hence the necessity to avoid mixed storage of lettuce with apples, pears or other climacteric fruits (7).



243 Tip-burn of witloof chicory



244 Bacterial soft rot of red-leaved chicory (radicchio)

Chicory, *Cichorium intybus* L., was originally grown mainly for its bitter roots, which (dried and ground) can be used as an additive or substitute for coffee. Chicory roots may also be used as a vegetable and have occasionally, and unjustifiably, been offered for sale under the name of 'white radish', to which chicory is quite unrelated. This confusion probably arises from the Italian 'radicchio' which refers to particular types of salad chicory grown for their delicate red leaves (6). These, together with green-leaved varieties, bear some resemblance to lettuces, but possess a characteristic bitter flavour.

The celebrated Belgian variety, known by its Flemish name of 'witloof' (white leaf) (5), is produced by growing the plants in the dark under carefully controlled conditions (8). The resulting pale, succulent, pointed heads ('chicons') (9) may be eaten raw or cooked. The crop is mainly grown in France, Belgium and the Netherlands, a substantial proportion being for export. Confusingly, the French name for chicory is 'endive' (9) and, for endive, 'chicorée frisée'.

Endive, *C. endivia* L. (4), possesses a loose head of leaves, which are often much divided and curled. Broad-leaved cultivars are sometimes distinguished by the name of escarole. Blanching the leaves during growth serves to prevent them from becoming excessively bitter.

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2 BRECHT J.K., SHERMAN M. & ALLEN J.J. (1986) Film wrapping to improve the postharvest quality of Florida head lettuce. *Proceedings of Florida State Horticultural Society* **99,** 135–140.
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6 LANGTON F.A., BADR H.M. & JONES S.K. (1988) In-vitro culture of radicchio (*Cichorium intybus* L.) – a photoessay. *Annual Report of the Glasshouse Crops Research Institute (UK) 1986–87,* pp. 126–134 illus.
7 PATTERSON C.L., GROGAN R.G. & CAMPBELL R.N. (1986) Economically important diseases of lettuce. *Plant Disease* **70,** 982–987.
8 POLL J.T.K. & DOUGLAS J.A. (1987) Chicory: an investigation of production for witloof in the Waikato, New Zealand. *New Zealand Journal of Experimental Agriculture* **15,** 61–65.
9 SOUDAIN P., COCHET J.P. & MARLE M. (1985) Problèmes posés par la conservation de l'endive. *Acta Horticulturae* No. 157, 237–244.
10 WANG C.Y., HINSCH R.T. & KINDYA W.G. (1984) Effect of different perforations in polyethylene films on quality of head lettuce after transcontinental shipment. *HortScience* **19,** 584–586.

BACTERIAL ROTS of lettuce, chicory and endive caused by

***Erwinia* spp.**
***Pseudomonas* spp.**
***Xanthomonas* spp.**

OCCURRENCE Various bacteria may cause rots, and their incidence is often related to locality or season of the year. Thus in Japan, for example, *E. carotovora* ssp. *carotovora* (page 302) is associated with the spring crop, *Pseudomonas cichorii* is generally confined to the summer crop, while *Pseudomonas marginalis* pv. *marginalis* and *P. viridiflava* (pages 142, 143) occur predominantly in the winter crop grown under cover but subject to frost or excessive humidity. *Xanthomonas campestris* pv. *vitians* (Brown) Dye occurs only in certain localities (7). Other records include *E. carotovora* ssp. *carotovora* on lettuce in the USA (10) and in Hawaii (3); *P. cichorii* on lettuce in the USA (5) and Italy (1); *P. marginalis* pv. *marginalis* on lettuce in the USA (2) and New Zealand (6) and on chicory in Belgium (8); and *X. campestris* pv. *vitians* on lettuce in the USA (2) and in South Africa (9).

SYMPTOMS *E. carotovora* ssp. *carotovora* causes discoloration of vascular tissue at the cut stem-end or butt, and characteristically gives rise to a soft rot in which affected tissue is reduced to a dark green slimy mass (3,10). *P. cichorii* can cause firm dark brown lesions, the disease on lettuce being known as 'varnish spot' (1,5). *P. marginalis* pv. *marginalis* is responsible for marginal leaf blight (6), vascular blackening, and also a slimy soft rot (4). *X. campestris* pv. *vitians* causes bacterial spot, characterised by minute circular watersoaked lesions which coalesce to form irregular areas up to 5 mm across (9).

BIOLOGY These bacteria survive in the soil, and are favoured by wet conditions (6). *X. campestris* pv. *vitians* can cause direct infection of intact leaves (9), while *P. marginalis* and *E. carotovora* characteristically invade via growth splits, mechanical injuries, or physiologically weakened tissue (4,10). At temperatures between 20° and 30°C bacterial rots proceed rapidly. *E. carotovora* makes little growth at 5°C and below, whereas species of *Pseudomonas* can cause serious rotting even at temperatures close to 0°C (4).

CONTROL Waterlogging of the soil should be avoided and measures should be taken to prevent physiological disorders such as tipburn (q.v.). Harvested plants should be carefully trimmed, rapidly cooled, and held at just above freezing point. Crops known to be infected with species of *Pseudomonas* should not be stored for long, since these bacteria are not fully controlled by refrigeration (4).

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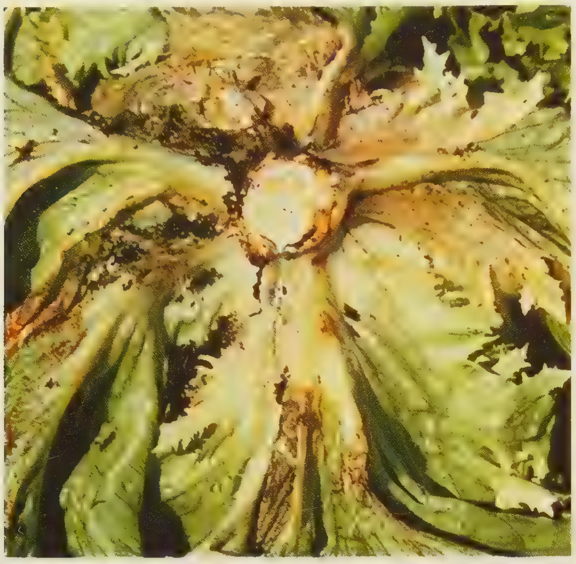
1 BAZZI C., PIAZZA C. & MAZZUCCHI U. (1984) Survival in the field of *Pseudomonas cichorii* (Swingle) Stapp, causal agent of lettuce varnish spot. *Phytopathologische Zeitschrift* **111**, 251–258.
2 BURKHOLDER W.H. (1954) Three bacteria pathogenic on head lettuce in New York State. *Phytopathology* **44**, 592–596 illus.
3 CHO J.J. (1977) Control of bacterial soft rot of crisphead type lettuce in Hawaii. *Plant Disease Reporter* **61**, 783–787 illus.
4 FRIEDMAN B.A. (1951) *Pseudomonas marginalis* as the cause of soft rot of imported Witloof chicory. *Phytopathology* **41**, 880–888 illus.
5 GROGAN R.G., MISAGHI I.J., KIMBLE K.A., GREATHEAD A.S., RIRIE D. & BARDIN R. (1977) Varnish spot, destructive disease of lettuce in California caused by *Pseudomonas cichorii*. *Phytopathology* **67**, 957–960.
6 MILLER S.A. (1980) Susceptibility of lettuce cultivars to marginal leaf blight caused by *Pseudomonas marginalis* (Brown 1918) Stevens 1925. *New Zealand Journal of Experimental Agriculture* **8**, 169–171.
7 OHATA K.-I., TSUCHIYA Y. & SHIRATA A. (1979) [Difference in kinds of pathogenic bacteria causing head rot of lettuce of different cropping types.] *Annals of the Phytopathological Society of Japan* **45**, 333–338.
8 OUTRYVE M.F. van, GOSSELE F., JOOS H. & SWINGS J. (1989) Fluorescent *Pseudomonas* isolates pathogenic on witloof chicory leaves. *Journal of Phytopathology* **125**, 247–256.
9 WALLIS F.M. & JOUBERT J.J. (1972) Bacterial leafspot of lettuce in Natal. *Phytophylactica* **4**, 137 illus.
10 WEHLBURG C. & MEYER R.W. (1966) Bacterial soft rot of Iceberg (Great Lakes) lettuce in the Florida Everglades. *Plant Disease Reporter* **50**, 938–941 illus.

BOTTOM ROT or **RHIZOCTONIA ROT** of lettuce caused by

***Thanatephorus cucumeris* (Frank) Donk**
Sclerotial state: *Rhizoctonia solani* Kühn

245



OCCURRENCE In Europe this disease appears to be of particular importance in lettuce grown under glass or plastic, whereas in North America it has also caused losses in outdoor crops. The problem has been studied in Austria (1), France (2), the Netherlands (3), the UK (6) and the USA (4,5). Bottom rot is a disease complex, often involving several fungi and bacteria (3,5,6).

SYMPTOMS Discrete sunken rust-coloured lesions may be present on the mid-ribs of outermost leaves. If decay has spread to the leaf blades, diseased tissue is either covered with visible mould growth or is slimy and dark, often as a result of invasion by secondary organisms (3,5,6).

BIOLOGY The fungus survives in the soil as sclerotia and resistant mould strands. Lettuce plants become vulnerable to attack as their outermost leaves come into contact with the soil surface. Fungal development is favoured by moist conditions and, more especially, by temperatures around 20°C, although there are some strains which are active below 10°C (3). Infection occurs through natural openings (stomata) or by direct penetration. Invaded leaves may become black 'leaf-prints' on the soil surface, the main veins being visible as thin threads (3). If conditions remain warm and humid, a head-rot ensues. Other lettuces may be fit for harvest but need excessive trimming and, if infected leaves remain, decay continues during transport and storage.

CONTROL Field hygiene is important and crop debris should be promptly removed. Soil disinfection may be feasible (2,3,4). Fungicides can be sprayed on the soil surface before the young plants have expanded (3). Special sprayers have been designed to apply fungicide to the basal portions of maturing plants, so that the undersides of the outermost leaves are protected (4). Raised beds can be advantageous (4). Excessive use of fertilizer is inadvisable, because luxuriant growth leads to lack of air circulation in the crop and the consequent humid microclimate favours infection. Some lettuce cultivars tend to escape disease by virtue of their upright growth habit (3). The more spreading types can be protected from contact with the soil by a polyethylene mulch (2). Biological control, by means of an antagonistic fungus, tends to be more successful in greenhouses than in the field (1).

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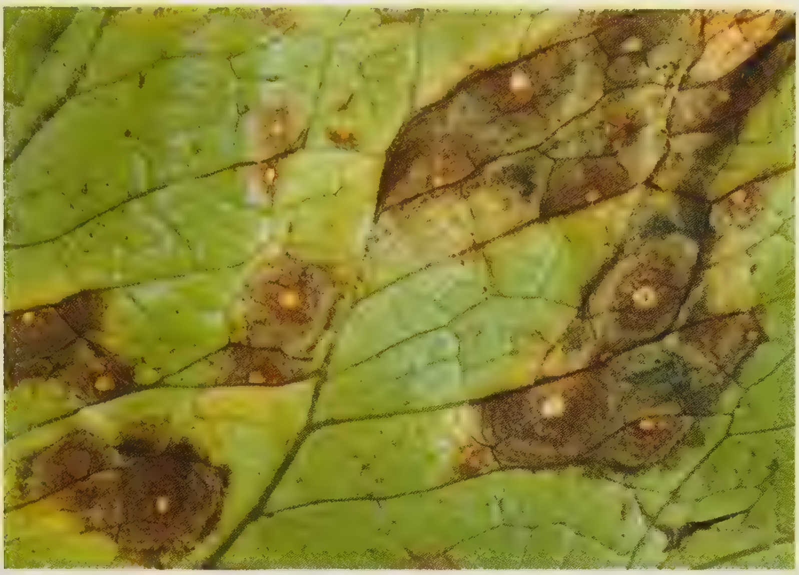
2 CAMPOROTA P., BAUDRAND M. & TAUSSIG C. (1986) Pourriture basale de la laitue provoquée par *Rhizoctonia solani*: essais de traitement au champ. *Revue Horticole* No. 271, 33–38.
3 KOOISTRA T. (1983) *Rhizoctonia solani* as a component in the bottom rot complex of glasshouse lettuce. *Mededelingen Plantenziektenkundige Dienst* No. 160, 144 pp.
4 MAHR S.E.R., STEVENSON W.R. & SEQUEIRA L. (1986) Control of bottom rot of head lettuce with iprodione. *Plant Disease* **70,** 506–509.
5 PIECZARKA D.J. & LORBEER J.W. (1975) Microorganisms associated with bottom rot of lettuce grown on organic soil in New York State. *Phytopathology* **65,** 16–21 illus.
6 WAREING P.W., WANG Z.-N., COLEY-SMITH J.R. & JEVES T.M. (1986) Fungal pathogens in rotted basal leaves of lettuce in Humberside and Lancashire with particular reference to *Rhizoctonia solani*. *Plant Pathology* **35,** 390–395.

CERCOSPORA SPOT of lettuce caused by
Cercospora longissima Cugini ex Traverso



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OCCURRENCE Cercospora spot of lettuce is found predominantly in tropical countries, for example Colombia, Honduras, Puerto Rico, the Philippines (1), Hong Kong (5), India (2), the Ivory Coast (4) and Somalia (3). It has also been recorded in Italy (2). There are also several brief accounts of various species of *Cercospora* on chicory.

SYMPTOMS Minute watersoaked brown spots, each surrounded by a pale halo, appear on the outer leaves of lettuce (5). The lesions enlarge into irregular brown areas bounded by the leaf veins, and coalesce to form large blotches. In a humid atmosphere there is a greyish to buff growth of mould giving rise to spores (conidia) (2,4). The disease may also spread to younger leaves (5).

BIOLOGY The fungus survives on crop debris in the soil, and its spores are disseminated by watersplash. Severe outbreaks of the disease are associated with the rainy season (4). The spores germinate on wet leaves, and cause infection via the natural openings (stomata) (4,5). Disease development and spore formation are favoured by temperatures between 25° and 35°C coupled with a saturated atmosphere (3,4,5).

CONTROL Crop rotation is helpful, as is destruction of infected crop debris (4). Disease incidence can be reduced by wider spacing of plants (which facilitates aeration and hence evaporation of moisture), and by application of fungicide sprays (3).

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1 McWHORTER F.P. (1925) Cercospora leaf spot of lettuce. *Phytopathology* **15,** 247.

2 MATHUR R.S., SINGH D.V. & PRASAD S.N. (1964) *Cercospora longissima* Cugini ex Traverso on lettuce – a new record for India. *Current Science* **33,** 624–625 illus.

3 MOHAMED I.M. (1979) Osservazioni sulla cercosporiosi della lattuga in Somalia. *Studi e Ricerche* No. **3,** 352–357.

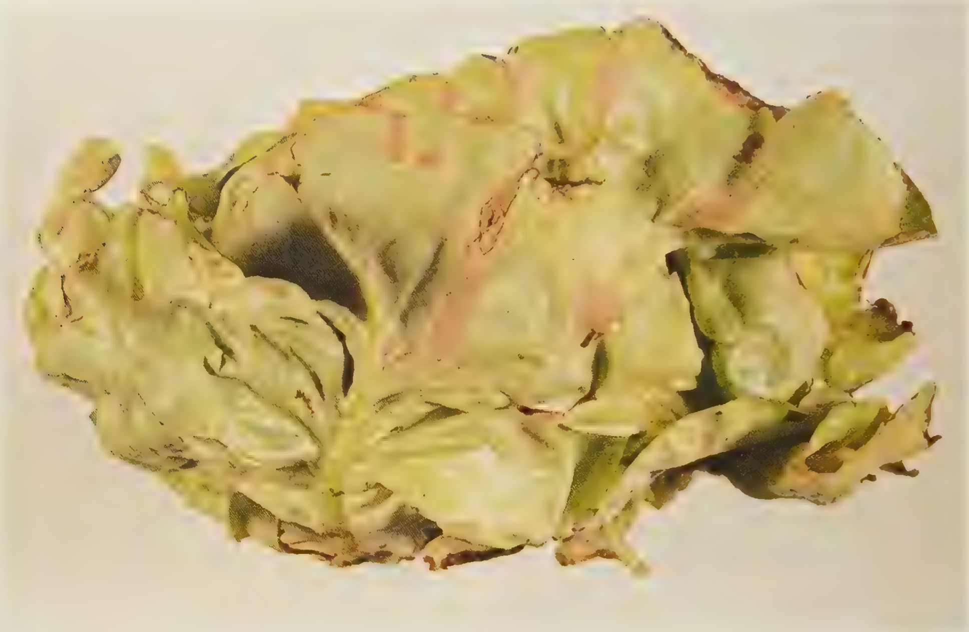
4 SAVARY S. (1983) Épidémiologie de la cercosporiose de la laitue (*Lactuca sativa* L.) en République de Côte d'Ivoire: étude de quelques étapes du cycle épidémiologique. *Agronomie* **3,** 903–909.

5 SZETO M. & BAU Y.S. (1975) Some notes on leaf spot (*Cercospora longissima*) disease of lettuce. *Agriculture Hong Kong* **1,** 278–285 illus.

6 WELLES C.G. (1923) Leaf-spot of lettuce. *Phytopathology* **13,** 289.

DOWNY MILDEW of lettuce, chicory and endive caused by ***Bremia lactucae*** **Regel**

247



OCCURRENCE Downy mildew is chiefly important in temperate regions such as the UK (1,3,7), France (6), the Netherlands (9), Czechoslovakia (4) and parts of the USSR. However, it has also been recorded throughout the USA, including the south (5), and in several tropical countries during their cool season (CMI Map 86). The disease is important in both field and greenhouse crops (9,10). Several races of the fungus exist; some forms infect lettuce (8), others endive and chicory.

SYMPTOMS Pale green or yellow areas develop on the leaves, the lesions being generally bounded by the veins and therefore angular in shape. Characteristic white spores (conidia) are produced on the underside or on both surfaces of the leaves (9).

BIOLOGY The fungus persists on crop debris (6) in the sexual state (oospores) (3,10). These spores germinate and may infect seedlings (10). Infected leaves then support abundant asexual spores (conidia) which are dispersed by air and impinge on healthy plants. Conidia germinate only if the leaves remain wet for some hours, whether from irrigation water, rain or dew (9). Infection can occur by direct penetration of the intact leaf surface, as well as via the natural openings (stomata) (5). Plants are especially prone to attack if their growth has been checked by adverse factors such as low temperature, poor light, temporarily dry soil, or virus infection. Downy mildew is essentially a disease of the growing crop, but slightly infected lettuce can undergo substantial further deterioration during transit or storage, especially in the event of secondary bacterial soft rot (q.v.) or grey mould rot (q.v.) (5,9).

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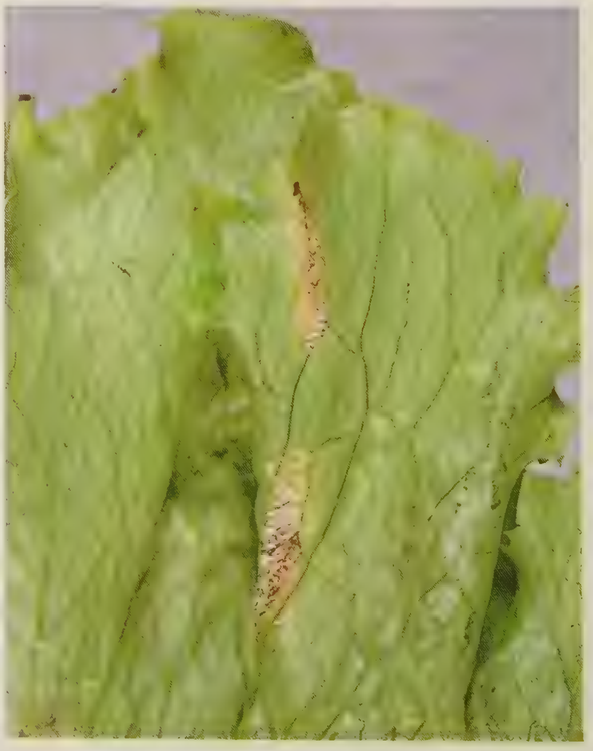
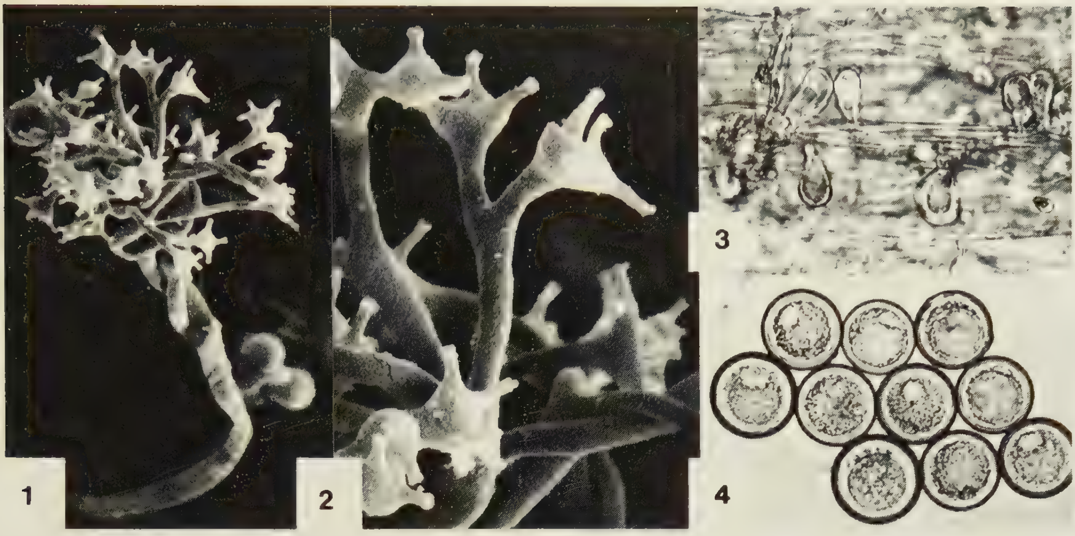




249 *Bremia lactucae* (from CMI Descr. No. 682, Morgan 1981) 1 conidiophore × 400 2 conidiophore, branch endings × 1300 3 haustoria × 400 4 oospores × 400

CONTROL A range of methods is called for (7). Successive sowings in the same vicinity should be avoided, as spores from one crop are likely to infect the next. Fungicide sprays should be applied as a preventive measure before the appearance of symptoms (1,7,9). Care should be taken to maintain good plant vigour by appropriate irrigation where necessary and, in greenhouses, by judicious use of ventilation and possibly supplementary heat (7). After harvest, debris should be collected, composted, and treated with a fungicide (6). Much effort has been put into breeding lettuce cultivars with resistance to various races of the fungus (2,4), but it is a continuing task since the fungus readily produces new races, including strains which are insensitive to current fungicides (1,4).

1 CRUTE I.R. & HARRISON J.M. (1988) Studies on the inheritance of resistance to metalaxyl in *Bremia lactucae* and on the stability and fitness of field isolates. *Plant Pathology* **37,** 231–250.

2 GUSTAFSSON I. (1989) Potential sources of resistance to lettuce downy mildew (*Bremia lactucae*) in different *Lactuca* species. *Euphytica* **40,** 227–232.

3 HUMPHREYS-JONES D.R. (1971) Studies on a method of carry-over of downy mildew (*Bremia lactucae*) of lettuce. *Plant and Soil* **35,** 187–188.

4 LEBEDA A. (1984) Race-specific factors of resistance to *Bremia lactucae* in the world assortment of lettuce. *Scientia Horticulturae* **22,** 23–32.

5 MARLATT R.B. (1974) Biology, morphology, taxonomy and disease relations of the fungus *Bremia*. *Bulletin of Florida Agricultural Experiment Station* No. 764, 25 pp.

6 MARTIN C. (1982) Les possibilités pratiques de traitement fongicide du terreau à propos du brémia et du sclérotinia de la laitue. *Revue Horticole* No. 228, 21–25.

7 MORGAN W.M. (1984) Integration of environmental and fungicidal control of *Bremia lactucae* in a glasshouse lettuce crop. *Crop Protection* **3,** 349–361.

8 SKIDMORE D.I. & INGRAM D.S. (1985) Conidial morphology and the specialization of *Bremia lactucae* Regel (Peronosporaceae) on hosts in the family Compositae. *Botanical Journal of the Linnean Society* **91,** 503–522.

9 VERHOEFF K. (1960) On the parasitism of *Bremia lactucae* Regel on lettuce. *Tijdschrift over Plantenziekten* **66,** 133–203 illus.

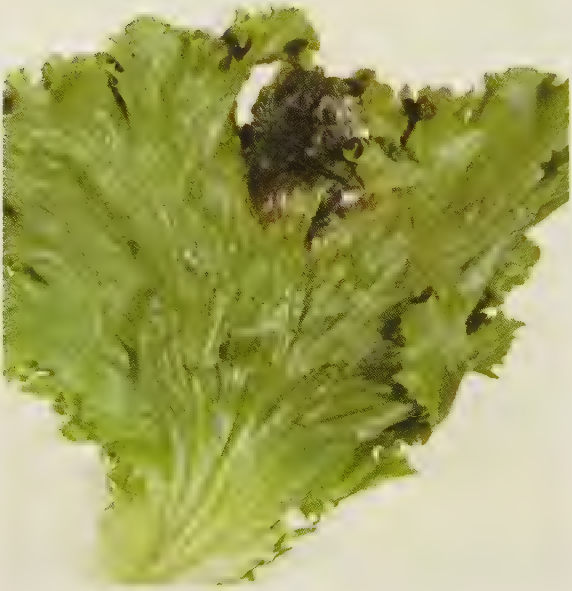
10 YUEN J.E. & LORBEER J.W. (1987) Natural and experimental production of oospores of *Bremia lactucae* in lettuce in New York. *Plant Disease* **71,** 63–64.

GREY MOULD ROT of lettuce and endive caused by

Botryotinia fuckeliana **(de Bary) Whetzel**

Conidial state: *Botrytis cinerea* Pers.

250



OCCURRENCE Grey mould rot occurs on lettuce in many countries, for example the USA (2,5), Canada, the UK (1,3), France (4), Germany, India, Australia and Japan. The disease also occurs on endive (4). Protected and outdoor crops may be attacked (3,4).

SYMPTOMS Brownish watersoaked lesions are found predominantly on outer leaves, especially at the margins, and a soft wet decay spreads through the head, sometimes associated with bacterial soft rot (q.v.) (2). Grey mould may also be a component of 'bottom rot' (q.v.). Under humid conditions there is profuse production of characteristic grey-brown spores (conidia); subsequently, black resting bodies (sclerotia, approximately 2 to 5 mm) may form in the decayed tissue. Strains of the fungus vary in the relative abundance of spores and sclerotia (1).

BIOLOGY The fungus persists in the soil as sclerotia or as mould strands in crop debris, and its spores are blown about in the atmosphere (page 281). Plants are predisposed to infection if they have been weakened in some way, for example through physical damage, low temperature, drought, sunburn, or the physiological disorder known as 'tipburn' (q.v.). In many localities there are well-defined fluctuations in disease incidence according to the time of year, grey mould being especially prevalent during cool humid seasons (1,2). Freshly trimmed plants may appear sound at harvest, but deterioration can occur during transit, even at temperatures close to 0°C (2).

CONTROL Crop rotation can be beneficial, and strict hygiene is advisable in field and greenhouse, including prompt disposal of crop debris (1). Care should be taken to prevent sudden checks in plant growth, by ensuring adequate irrigation of the crop. Greenhouses should be suitably ventilated (and heated if necessary) in order to prevent condensation on the plants. Sprinkler irrigation and spraying should be carried out early in the day so as to allow time for thorough drying of foliage before nightfall. New fungicides are being introduced to control strains of grey mould which have developed a tolerance to chemicals formerly effective against them (3,4). The harvested crop should be carefully trimmed and cooled without delay, since low temperatures help to minimise the spread of grey mould rot. Controlled atmosphere storage (reduced oxygen and enhanced carbon dioxide) can inhibit development of grey mould (5).

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1 ABDEL-SALAM M.M. (1934) Botrytis disease of lettuce. *Journal of Pomology and Horticultural Science* **12,** 15–35 illus.

2 CEPONIS M.J., KAUFMAN J. & BUTTERFIELD J.E. (1970) Relative importance of gray mold rot and bacterial soft rot of western lettuce on the New York market. *Plant Disease Reporter* **54,** 263–265.

3 CHANNON A.G., STEWART D.J. & HALBERT D. (1981) Effect of HV and ULV applications of fungicides on the control of grey mould (*Botrytis cinerea* Pers.) and the yield of glasshouse lettuce. *Horticultural Research* **21,** 159–168.

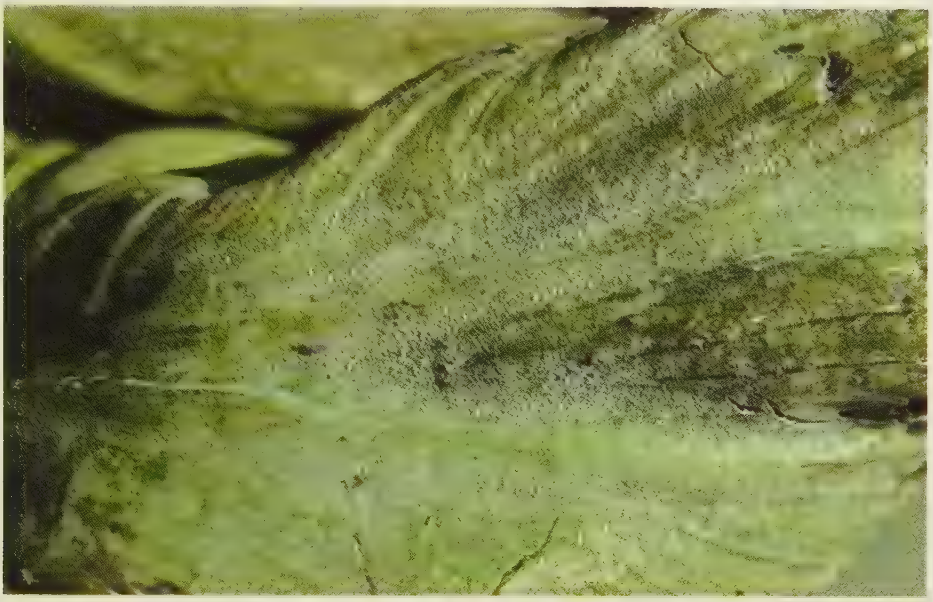
4 MARCHAND D. (1979) La lutte en végétation contre les pourritures à *Botrytis* et *Sclérotinia* des laitues et chicorées à feuilles. *Revue Horticole* No. 198, 49–51.

5 STEWART J.K. (1978) Influence of oxygen, carbon dioxide and carbon monoxide levels on decay of head lettuce after harvest. *Scientia Horticulturae* **9,** 207–213.

POWDERY MILDEW of lettuce, chicory and endive caused by

***Erysiphe cichoracearum* f.sp. *lactucae* DC. ex Merat**

Conidial state: *Oidium* sp.



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OCCURRENCE Powdery mildew has been recorded on lettuce in the western USA (2,5), Canada, the UK (1,3) and several other European countries (4). Crops grown in field or greenhouse may be attacked. The disease also affects chicory and endive (2).

SYMPTOMS Lesions develop on the outer leaves, first on the undersurface and later on both surfaces (1). There is profuse production of powdery white spores (conidia). Severely infected leaves become dry and discoloured as if they had been scorched, and give off a distinctive odour. Minute black bodies (cleistothecia) may develop near the midrib or near large veins.

BIOLOGY Originally this fungus attacked only wild lettuce (*Lactuca serriola* L.), but by the 1950s a strain had evolved which was capable of infecting cultivated lettuce (2,5). The sexual stage (cleistothecia giving rise to ascospores) has been recorded both on lettuce plants and on weeds (2,4). During mild wet weather ascospores are released and cause primary infections in the developing crop. Subsequent spread of the disease is by asexual spores (conidia) which form on the foliage of infected plants and are widely dispersed by wind (5). In contrast to downy mildew (q.v.), powdery mildew is severe at moderate temperatures (optimum 18°C) and moderate relative humidities (5).

CONTROL Some lettuce cultivars are resistant to attack by the powdery mildew fungus (3,4). Where susceptible cultivars are grown, strict quarantine measures may be enforced to prevent the introduction of virulent strains of the fungus (1).

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1 CRUTE I.R. & BURNS I.G. (1983) Powdery mildew of lettuce (*Lactuca sativa*). *Plant Pathology* **32**, 455–457 illus.

2 DESLANDES J.A. (1954) Studies and observations on lettuce powdery mildew. *Plant Disease Reporter* **38**, 560–562.

3 KNIGHT C., HOLLAND R.W.K. & WRIGHT I.R. (1986) Resistance of lettuce varieties to powdery mildew. *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108**, Supplement), 150–151.

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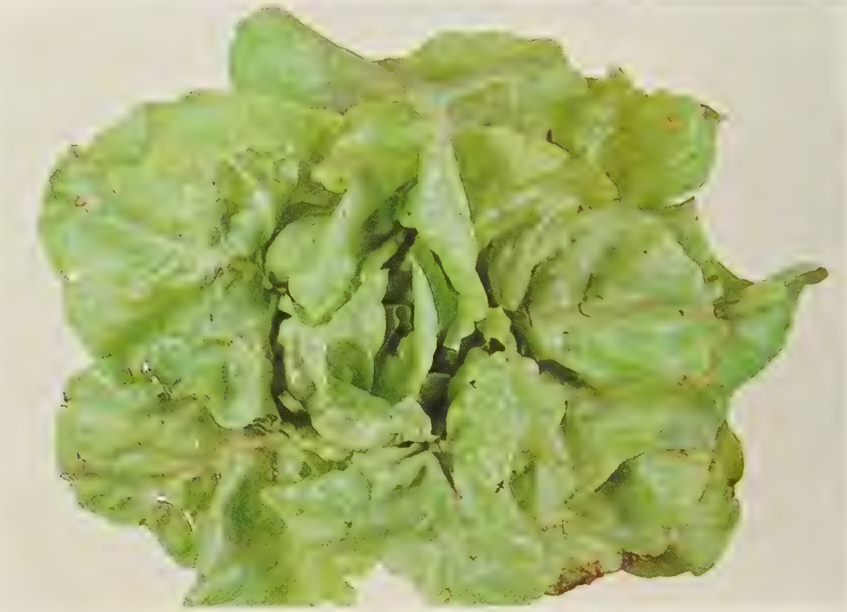
RINGSPOT or **ANTHRACNOSE** of lettuce, chicory and endive caused by ***Microdochium panattonianum*** **(Berl.) Sutton *et al.***

(syn. *Marssonina panattoniana* (Berl.) Magn.)

252



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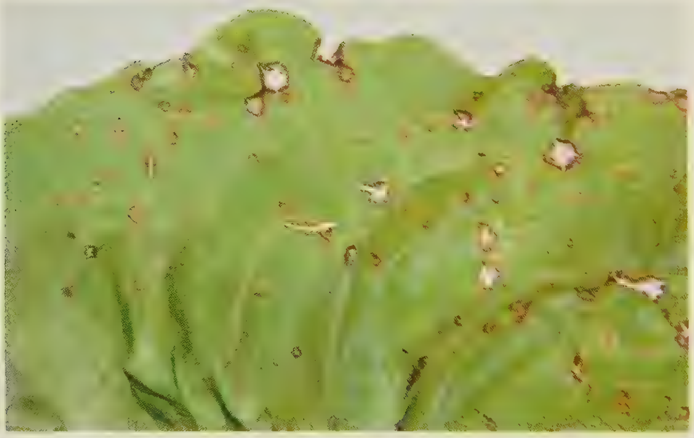
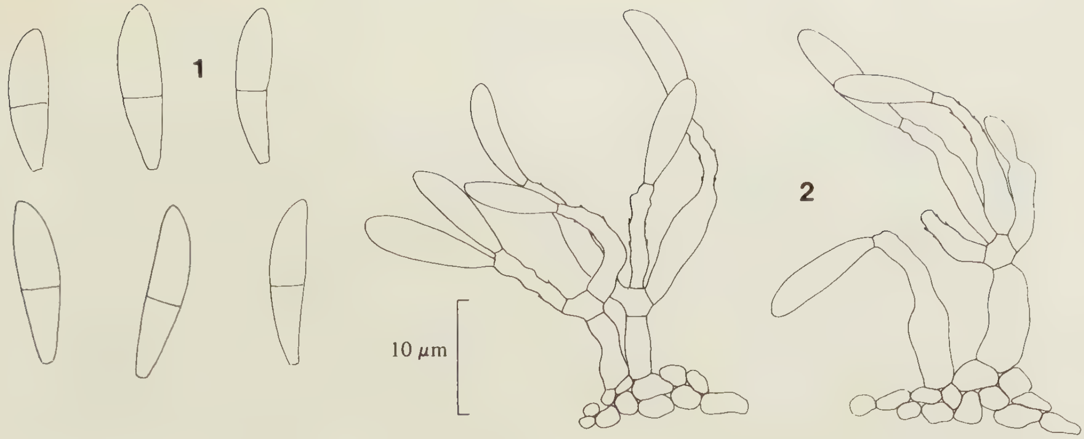


OCCURRENCE This disease is widespread in temperate growing areas, and may occur in the greenhouse (1) or in the field (6,7). It occasionally causes heavy losses in lettuce, for example in the USA (1,2,7,9), the UK (10), the Netherlands, Germany, Sweden, Hungary, Italy, Australia (4) and New Zealand (5). Chicory and endive may also be affected (1,2,4).

SYMPTOMS Ringspot of lettuce has been confused with stemphylium spot (q.v.) but can be distinguished by microscopic examination (5). Leaf spots (first apparent on the undersurface) are initially watersoaked and circular, developing rapidly into yellowish lesions (3 to 4 mm across) which, if bounded by large leaf veins, may be angular in shape (1,4). Subsequently the centres of the spots dry up and may fray or fall out, giving the leaf a characteristic shot-hole appearance (1,5). Lesions on the midrib also start as watersoaked spots but later become oval, reddish yellow, and markedly sunken (10). Moist conditions encourage the development of white or pink spores (conidia), especially on lesions along the midrib (7). According to early reports, the spores are produced in specialised structures (acervuli) (1,2). However, a more recent study with the aid of a scanning electron microscope reveals that this is not so, and suggests that it is therefore incorrect to classify this organism as an anthracnose fungus (7).



254 *Microdochium panattonianum* (from *Trans. Br. mycol. Soc.* **86,** 619–628, Galea, Price & Sutton 1986)
1 conidia 2 conidiophores and developing conidia

BIOLOGY The fungus survives in soil and on crop debris as microsclerotia (9) or else as mould strands in infected weeds such as wild lettuce (2,4). It may also be transmitted by seed (5,10). Wet weather or irrigation is conducive to the production of spores, which are dispersed by watersplash or wind-blown rain and serve to spread the disease from plant to plant (4). Infection occurs by direct penetration (3) or via natural openings (stomata) in the leaf tissue (2,3), and resulting lesions may render the plants unfit for harvest (4). Alternatively, if infection occurs shortly before harvest, the plants appear healthy when packed but deteriorate during storage or transit (7). Decay can spread into sound produce and, while the optimal temperature for disease development is 18°C, symptoms may eventually be manifest even at temperatures around 2°C (7).

CONTROL Since the fungus does not attack other crops (2), rotation is feasible as a means of control (1). It is important to sow clean seed (4,10) and to control weeds (4,5). It is inadvisable to move through the crop when the foliage is wet; furthermore, irrigation at ground level is preferable to sprinklers (1), although the latter may be used if timed correctly so as to minimise periods of leaf wetness. Where there is a history of the disease it is necessary to apply preventive fungicide sprays (6), using weather data as a guide, before the appearance of symptoms (5). Any plants showing signs of the disease should be promptly removed and burned (1), and crop debris should be destroyed at the end of the season (4,10). Work is in progress to breed resistant cultivars (8).

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1 BRANDES E.W. (1918) Anthracnose of lettuce caused by *Marssonina panattoniana. Journal of Agricultural Research* **13,** 261–280 illus.
2 COUCH H.B. & GROGAN R.G. (1955) Etiology of lettuce anthracnose and host range of the pathogen. *Phytopathology* **45,** 375–380 illus.
3 GALEA V.J. & PRICE T.V. (1988) Infection of lettuce by *Microdochium panattonianum. Transactions of the British Mycological Society* **91,** 419–425 illus.
4 HARRISON D.E. (1961) Anthracnose of lettuce. *Journal of the Department of Agriculture, Victoria* **59,** 354–356 illus.
5 HURNDELL L.C. & SMITH H.C. (1958) Ringspot disease of lettuce. *New Zealand Journal of Agriculture* **96,** 575–576 illus.
6 JONES D.R. (1986) Evaluation of fungicides for control of ringspot (*Marssonina panattoniana*) of outdoor lettuce. *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108,** Supplement) 56–57.
7 MOLINE H.E. & POLLACK F.G. (1976) Conidiogenesis of *Marssonina panattoniana* and its potential as a serious postharvest pathogen of lettuce. *Phytopathology* **66,** 669–674 illus.
8 OCHOA O., DELP B. & MICHELMORE R.W. (1987) Resistance in *Lactuca* spp. to *Microdochium panattoniana* (lettuce anthracnose). *Euphytica* **36,** 609–614.
9 PATTERSON C.L. & GROGAN R.G. (1985) The source and survival of primary inoculum produced by *Marssonina panattoniana,* the causal agent of lettuce anthracnose. *Phytopathology* **75,** 1319 [Abstract].
10 STEVENSON G.B. (1939) On the occurrence and spread of the ringspot disease of lettuce caused by *Marssonina panattoniana* (Berl.) Magn. *Journal of Pomology and Horticultural Science* **17,** 27–50.

SEPTORIA SPOT of lettuce caused by

Septoria lactucae **Pass.**

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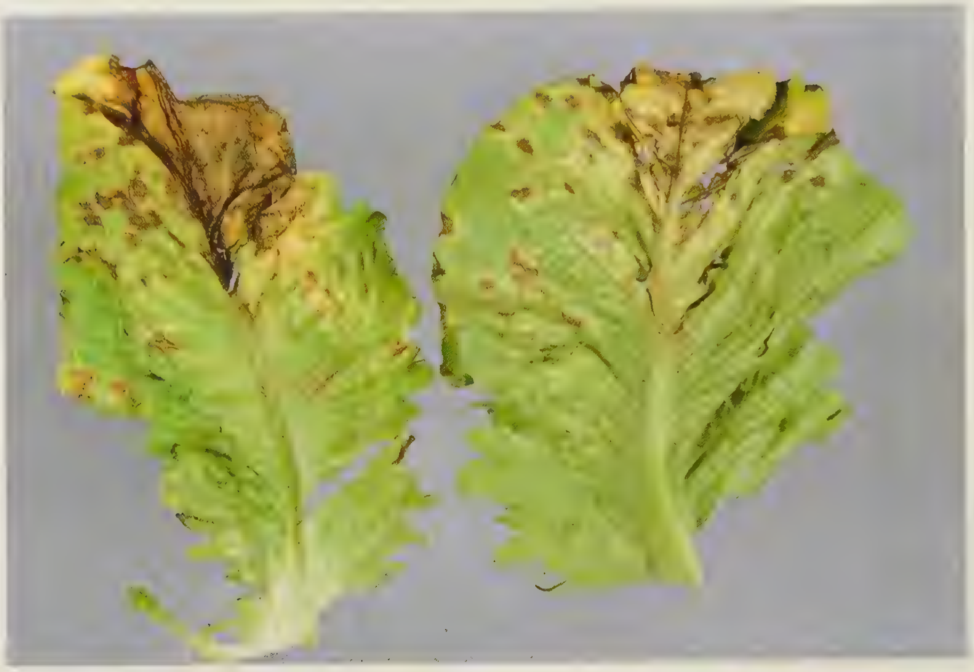

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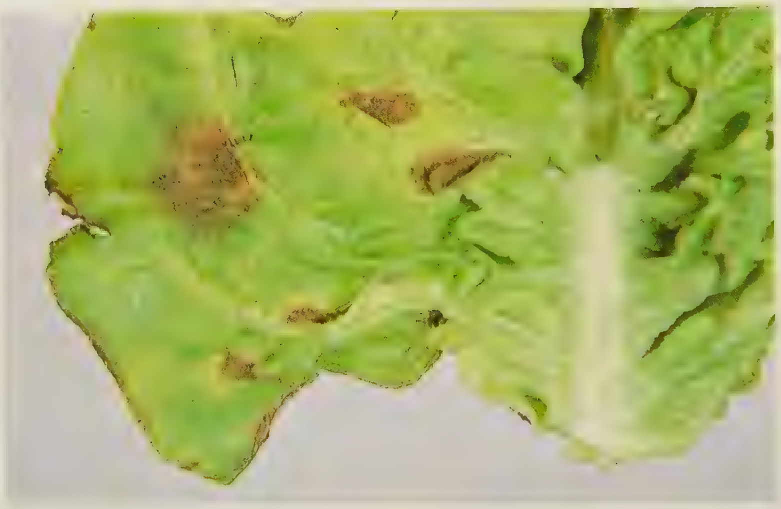
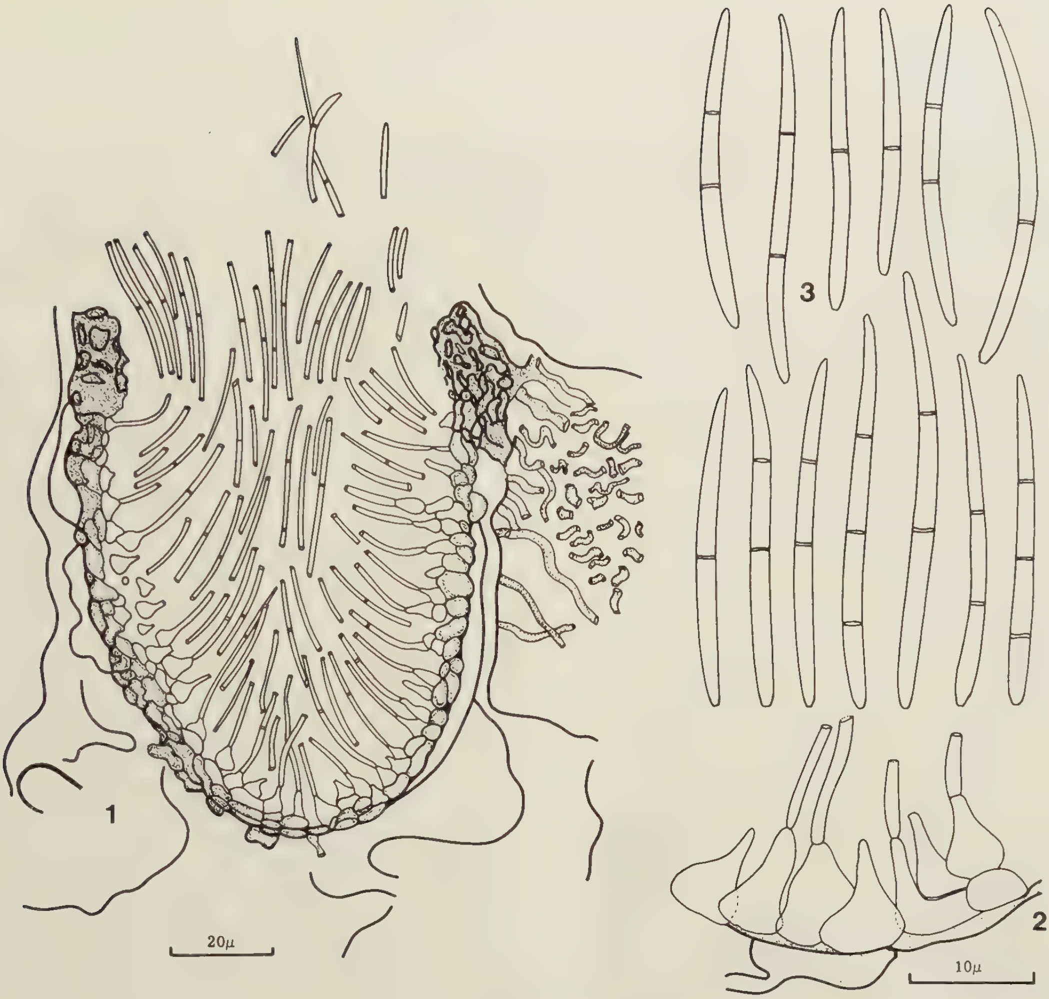


OCCURRENCE The fungus is widely distributed (CMI Map 485) and has caused losses in Australia (1,6), South Africa, Argentina (4), the USA, the West Indies (3), Sri Lanka (2) and the UK (5).

SYMPTOMS Irregularly shaped light brown or silvery spots appear on the outer leaves, and minute black bodies (pycnidia) develop within the affected areas on both surfaces of the leaf (1,2,6). The lesions coalesce, and the leaves become dry and withered.

BIOLOGY The fungus exists in the asexual state (pycnidia giving rise to conidia) and survives in weeds (2), on crop debris or on lettuce seed (3,5,6). During warm wet weather spores are disseminated by watersplash, and can cause infection of young plants (2). Older plants are more resistant, but if their growth is checked by adverse conditions (such as insufficient fertilization or excessive shading) they become susceptible to attack (1). Thus mature heads may be unfit for market and, in seed crops, the fungus can progress up the flowering stalk and infect flowers and seeds (6).



257 *Septoria lactucae* (from CMI Descr. No. 335, Punithalingam & Holliday 1972) 1 pycnidium 2 part of pycnidial wall and conidiophores 3 conidia

CONTROL If clean seed is not available, it is necessary to employ hot-water treatment to kill the fungus (6). However, this can lead to reduced seed-germination. The seed-bed should be disinfected (3), and a fungicide spray may be applied to the developing crop. Debris should be promptly removed from the field at the end of the season (3).

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1 BLACKFORD F.W. (1944) Downy mildew and Septoria leaf spot of lettuce. *Queensland Agricultural Journal* **59,** 221–223 illus.

2 BOND T.E.T. (1941) Leaf spot diseases of lettuce and antirrhinum. *Tropical Agriculturist* **97,** 62–67 illus.

3 FOURNET J. (1976) Possibilités d'amélioration de la lutte contre la septoriose de la laitue aux Antilles par l'étude des épidémies. *Annales de Phytopathologie* **8,** 41–50.

4 MITIDIERI I.Z.M. de (1977) Mancha de la hoja de la lechuga causada por *Septoria lactucae* Pass. *Idia* Nos. 355/360, 8–12.

5 MOORE W.C. (1940) New and interesting plant diseases. 8. Leafspot of lettuce (*Septoria lactucae* Pass.). *Transactions of the British Mycological Society* **24,** 345–351.

6 SMITH P.R. (1961) Seed-borne *Septoria* in lettuce. Eradication by hot water treatment. *Journal of the Department of Agriculture, Victoria* **59,** 555–556 illus.

STEMPHYLIUM SPOT of lettuce caused by
***Pleospora herbarum* f.sp. *lactucum* (Pers.) Rabenh.**

Conidial state: *Stemphylium herbarum* Simmons

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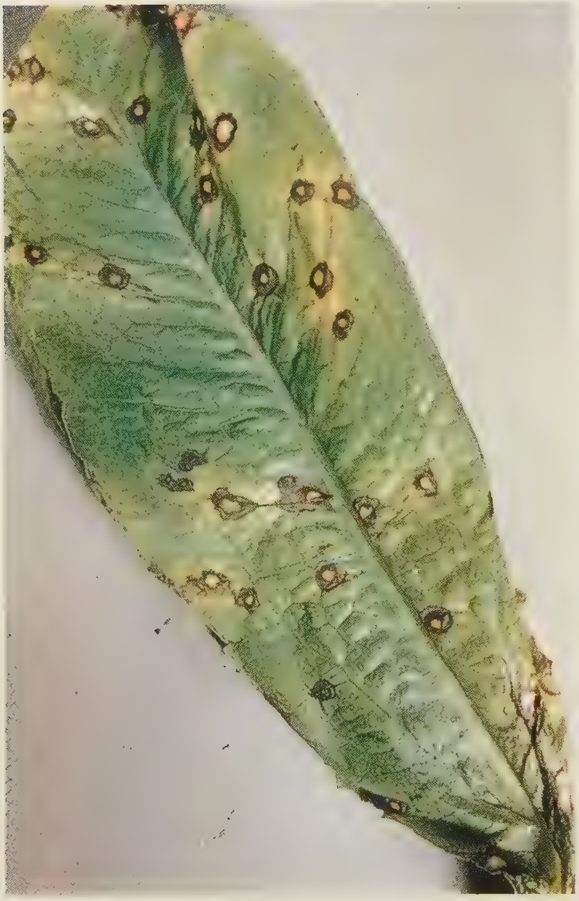

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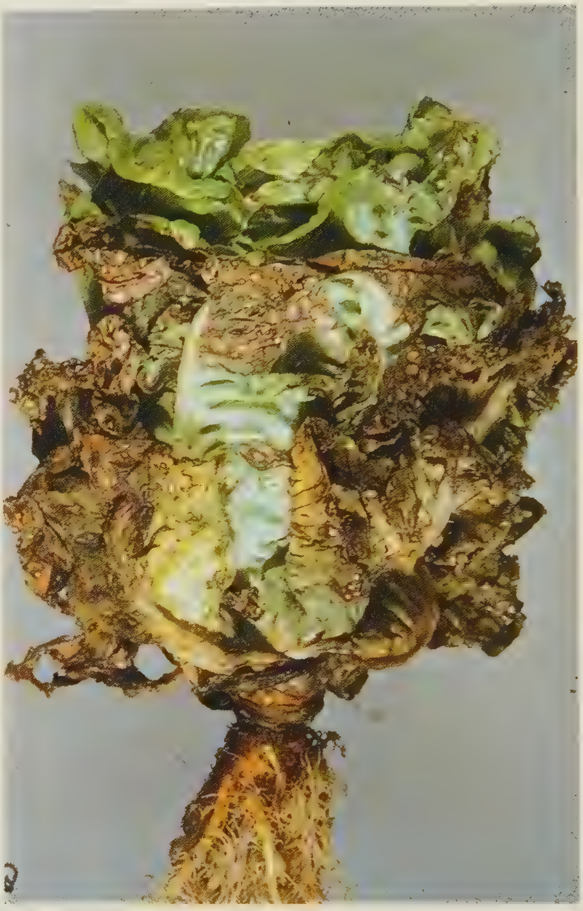
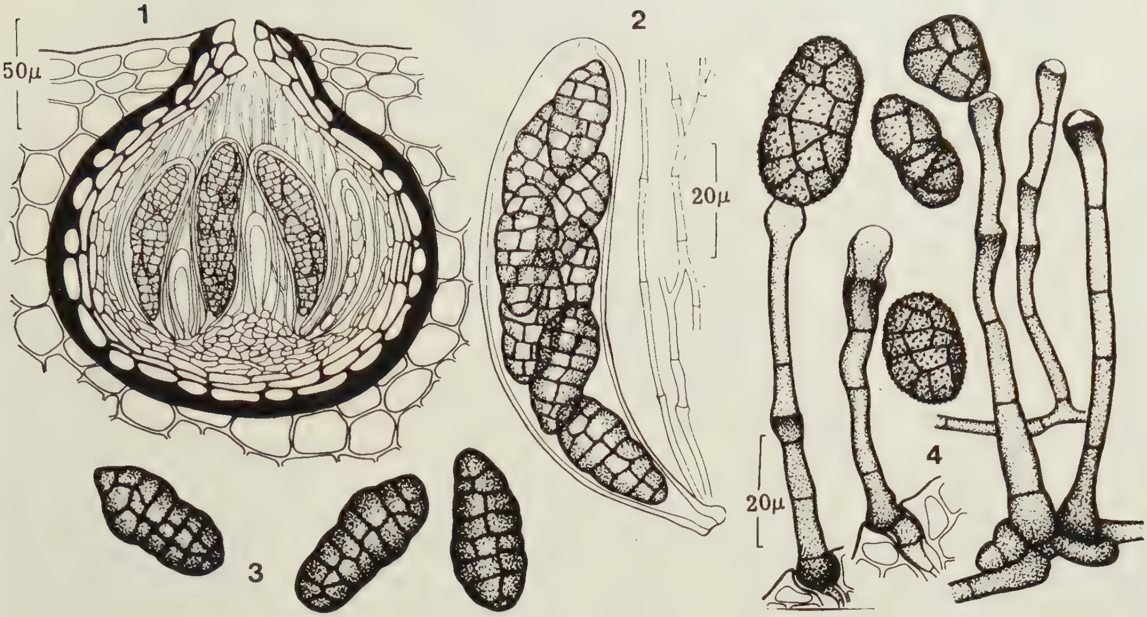


OCCURRENCE This disease has been recorded on lettuce in the UK, the Netherlands (2), Italy (6), Spain (1), Portugal, Israel (4,9), South Africa, New Zealand (10), the western USA (5) and Argentina (7).

SYMPTOMS Small watersoaked depressions appear on both upper and lower surfaces of the outermost leaf blades and midribs (10). The developing lesions may be circular, oval, or angular if bounded by the leaf veins, and they often show concentric light and dark zones, giving the impression of a target spot (2). The centres of the lesions may split or fall out, resulting in a shot-hole effect, and the disease has often been confused with ringspot (q.v.) (10). However, under humid conditions there is copious production of dark spores (conidia) (6).

BIOLOGY The fungus, which may be seed-transmitted (7), can survive in infected weeds (10). It persists in the sexual state (pseudothecia giving rise to ascospores) (3,8) on crop debris (5), or as asexual spores (conidia) which can withstand periods of hot dry weather (10). In cool wet seasons spores which are blown or splashed on to lettuce plants cause infection via natural openings (stomata) in the leaves (5). Blemished outer leaves can be trimmed off at harvest time, but unnoticed incipient infections continue to develop, causing serious losses even in refrigerated storage (5,9).

CONTROL Suggested methods of control include the use of resistant lettuce cultivars (4), the sowing of clean seed in disinfected soil, and the application of sprays or dusts before the disease becomes established in the crop (4,7,10). At the end of the season infected crop debris should be destroyed (5).



260 *Pleospora herbarum* (from CMI Descr. No. 150, Booth & Pirozynski 1967) 1 pseudothecium 2 ascus 3 ascospores 4 conidia and conidiophores

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1 ALFARO GARCÍA A. (1971) Una enfermedad de las lechugas malagueñas debida al *Stemphylium botryosum* Wallr. *Anales del Instituto Nacional de Investigaciones Agrarias (Ser. Protección Vegetal)* **1,** 63–69 illus.

2 GOOSSENS J. (1951) Een bladvlekkenziekte op jonge slaplanten veroorzaakt door *Pleospora herbarum* (Pers.) Rabenh. *Tijdschrift over Plantenziekten* **57,** 170–171 illus.

3 LEACH C.M. (1971) Regulation of perithecium development and maturation in *Pleospora herbarum* by light and temperature. *Transactions of the British Mycological Society* **57,** 295–315.

4 NETZER D., GLOBERSON D., WEINTAL C. & ELYASSI R. (1985) Sources and inheritance of resistance to Stemphylium leaf spot of lettuce. *Euphytica* **34,** 393–396.

5 PADHI B. & SNYDER W.C. (1954) Stemphylium leaf spot of lettuce. *Phytopathology* **44,** 175–180 illus.

6 PETTINARI C. (1951) *Stemphylium botryosum* Wallr. su foglie di lattuga. *Bollettino della Stazione di Patologia Vegetale, Roma, Ser. 3,* **9,** 31–40 illus.

7 SARASOLA M.A.R. de & SARASOLA A.A. (1957) Una enfermedad de la lechuga producida por *Stemphylium botryosum* en la Argentina. *Revista de la Facultad de Agronomía, La Plata* **33,** 83–91 illus.

8 SIMMONS E.G. (1985) Perfect states of *Stemphylium. 2. Sydowia* **38,** 284–293 illus.

9 SIVAN J. & BARKAI-GOLAN R. (1976) *Stemphylium botryosum* f. *lactucae* – its development *in vitro* and its pathogenicity to lettuce leaves. *Annals of Applied Biology* **82,** 425–432.

10 SLADE D.A. (1963) Leafspot disease of lettuce. *New Zealand Journal of Agriculture* **107,** 73–74 illus.

WATERY SOFT ROT of lettuce, chicory and endive caused by

Sclerotinia minor **Jagger**
Sclerotinia sclerotiorum **(Lib.) de Bary**

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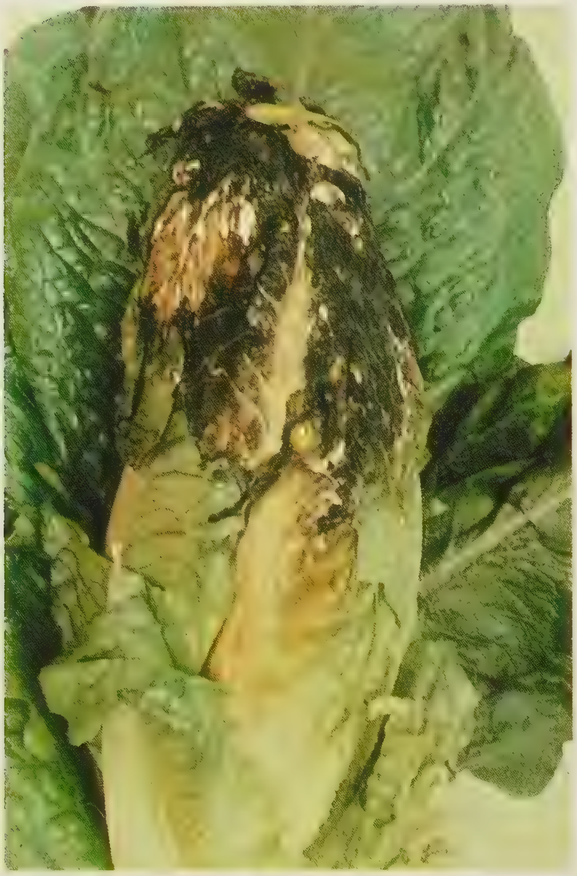
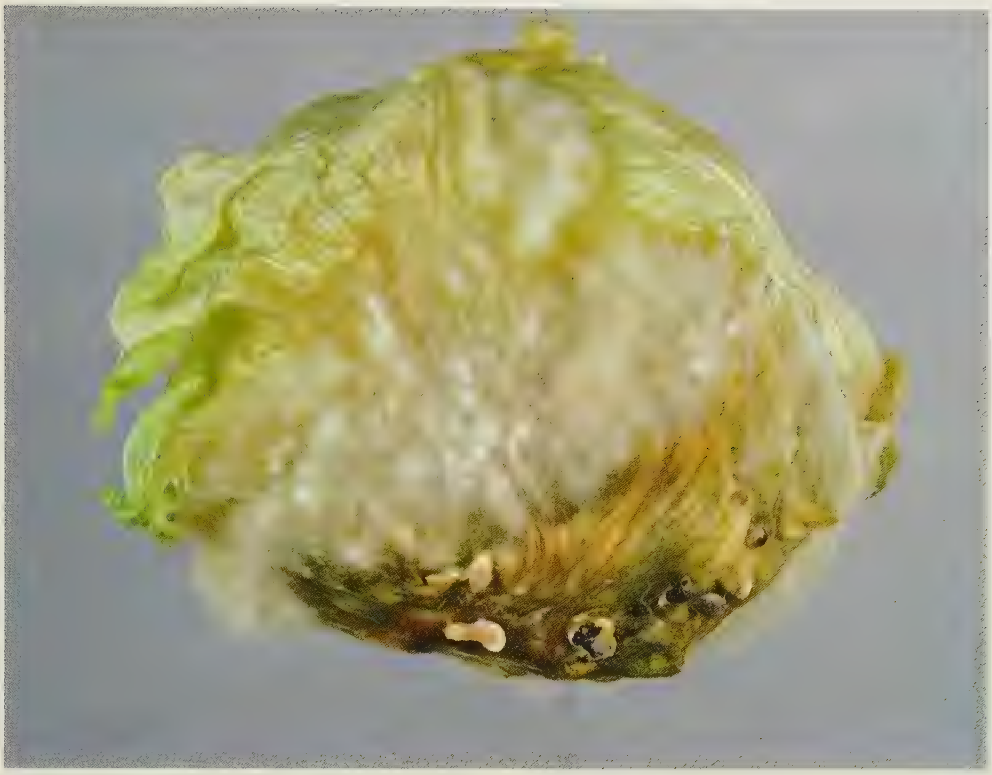


OCCURRENCE The disease is widespread in temperate growing areas, for example the UK (5), France (3), Germany (10), Israel (2), Australia (8), New Zealand (4), Canada and parts of the USA (1,6,9). It also occurs in subtropical regions possessing a cool wet season. Both field and greenhouse crops may be affected.

SYMPTOMS Infected tissue is watersoaked, pinkish brown, and later covered with a white cottony mould. As decay progresses there is copious leakage of juices, though no particular odour. Eventually resting bodies (sclerotia) form on and in the tissue; those of *S. minor* up to 2 mm, those of *S. sclerotiorum* up to 1 cm or more, irregular in shape.

BIOLOGY The two fungi may co-exist in the same field, but usually one or other predominates in a given locality (4,6). Mild wet weather favours development of both species but, in most instances, they have contrasting modes of growth and infection (9). The small sclerotia which are characteristic of *S. minor* typically produce a dense mass of mould strands, which infect outer leaves in contact with the soil (4) or else attack the root several cm below ground level (9). The large sclerotia which are characteristic of *S. sclerotiorum* require a more prolonged wet period, and those at or near the soil surface (8) germinate to produce the sexual stage (apothecia giving rise to ascospores). Spores are ejected into the air and, in the presence of moisture, can cause infection of leaves or stem, wherever there happens to be some dead or dying plant tissue. (Sclerotia of *S. minor* have been known to produce the sexual stage but this is rare in nature; the two fungi are generally regarded as distinct species.) Disease development in the field proceeds rapidly, and can lead to collapse of entire plants, hence the common name of 'lettuce drop' (1,9). Plants harvested with incipient infection undergo rotting in transit or storage, even under refrigeration, and decay spreads into adjacent sound produce.

CONTROL A range of measures is usually necessary, and it is particularly difficult to control *S. sclerotiorum* (2,8,9). The feasibility of crop rotation is limited by the wide host range of these fungi. Deep ploughing has been advocated as a means of burying sclerotia; however, some may remain viable and be turned up during subsequent cultivation (8). A surer way of killing sclerotia is to flood the land for a few weeks or, in places with a hot dry season, to cover the field temporarily with polyethylene sheeting in order to raise the soil temperature to a lethal level (solarization) (2). Alternatively it may be feasible to disinfect the soil with steam or chemicals before planting (2,5,10). The choice of cultivar is important; plants with an upright habit are more likely to escape the disease than are squat plants the outer leaves of which lie on the soil surface (4). If the latter types are grown, good control (particularly of *S. minor*) may be achieved by the use of a plastic mulch to prevent contact with the soil (4). Fungicide sprays may be applied (5,9,10) but it is difficult to ensure coverage of the basal region, which is particularly vulnerable to infection; systemic chemicals are the most effective. A fungicide lethal to sclerotia may be applied via the irrigation system (2). Constructing greenhouses from a special vinyl film can help to control *S. sclerotiorum* (by inhibiting development of apothecia) (page 135). If growing plants develop symptoms they should immediately be carefully removed and destroyed before more sclerotia can



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form (9). Harvested plants should be closely trimmed and rapidly cooled. After harvest it is sometimes customary to allow animals to graze on crop debris; most sclerotia are killed by passage through the digestive tract, but some may survive and be carried to new fields (7). Eventually, especially after alternating wet and dry periods, sclerotia undergo natural decay by other soil fungi, and efforts are being made to exploit possibilities of biological control using these organisms (1,3).

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1 ADAMS P.B. (1987) Effects of soil temperature, moisture, and depth on survival and activity of *Sclerotinia minor, Sclerotium cepivorum* and *Sporidesmium sclerotivorum. Plant Disease* **71,** 170–174.

2 BEN-YEPHET Y. (1988) Control of sclerotia and apothecia of *Sclerotinia sclerotiorum* by metham-sodium, methyl bromide and soil solarization. *Crop Protection* **7,** 25–27.

3 DAVET P. & MARTIN C. (1985) Effets de traitements fongicides aux imides cycliques sur les populations de *Sclerotinia minor* dans le sol. *Phytopathologische Zeitschrift* **112,** 7–16.

4 HAWTHORNE B.T. (1974) *Sclerotinia minor* on lettuce: effect of plant growth on susceptibility to infection. *New Zealand Journal of Agricultural Research* **17,** 387–392.

5 JONES D.R. (1986) Evaluation of fungicides for control of *Sclerotinia minor* on outdoor lettuce. *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108,** Supplement), 58–59.

6 MATHERON M.E. & MATEJKA J.C. (1989) In vitro and field comparison of six new fungicides with iprodione and vinclozolin for control of leaf drop of lettuce caused by *Sclerotinia sclerotiorum. Plant Disease* **73,** 727–730.

7 MELOUK H.A., SINGLETON L.L., OWENS F.N. & AKEM C.N. (1989) Viability of sclerotia of *S. minor* after passage through the digestive tract of a crossbred heifer. *Plant Disease* **73,** 68–69.

8 MERRIMAN P.P., PYWELL M., HARRISON G. & NANCARROW J. (1979) Survival of sclerotia of *Sclerotinia sclerotiorum* and effects of cultivation practices on disease. *Soil Biology and Biochemistry* **11,** 567–570.

9 PATTERSON C.L. & GROGAN R.G. (1985) Differences in epidemiology and control of lettuce drop caused by *Sclerotinia minor* and *S. sclerotiorum. Plant Disease* **69,** 766–770.

10 SCHMIDT H. (1965) Untersuchungen über die durch *Sclerotinia minor* Jagg. und *Botrytis cinerea* Pers. verursachten Salatfäulen. *Archiv für Pflanzenschutz* **1,** 179–207 illus.

OTHER DISEASES of lettuce, chicory and endive

ALTERNARIA SPOT, caused by various species of *Alternaria*, has been recorded on lettuce in several countries, on chicory in India, and on endive in Greece, Italy and Argentina (7). Endive cultivars are generally much more susceptible than chicory to infection by *A. cichorii* Nattrass (3).

MYCOCENTROSPORA SPOT has been recorded on outdoor lettuce in France (6) and in the UK, following heavy rains (5), and the disease also occurs in the USA. The causal fungus is *Mycocentrospora acerina* (page 285). Leaf spots are circular to angular, with pale brown centres and darker margins, sometimes surrounded by a broad band of greyish-green. Irregularly shaped lesions develop on the midribs of exposed leaves. Control by fungicide sprays has been suggested (5,6).

MYROTHECIUM ROT, caused by *Myrothecium roridum* (page 83), has been recorded on outdoor lettuce in Spain (9). Warm moist weather is conducive to infection, resulting in the formation of circular spots on both sides of the leaf blades (9).

PHYTOPHTHORA ROT has been recorded in Australia on lettuce, as a firm dark stem-rot extending upwards into the head (8). The disease was found only during winter, in badly drained fields. The causal fungus was a strain of *Phytophthora porri* (page 233) which had an optimal growth temperature of 15°C and failed to infect leeks or cabbages (8).

PYTHIUM ROT, caused by *Pythium aphanidermatum* (page 31), affects lettuces in India; leaves in contact with the ground are invaded, become water-soaked and decay rapidly. In the Netherlands species of *Pythium* are recorded as components of 'bottom rot' (q.v.).

RUST of lettuce occurs occasionally in the USA and parts of Europe, including Germany (4), the Netherlands and the UK (2). Lesions are especially noticeable on the undersurface of leaves, being reddish in colour and studded with pustules. Various species of *Puccinia* have been recorded in different countries; some complete their life cycle on lettuce, while others, for example *Puccinia opizii* Bubák (2,4), have two alternate hosts, producing spermagonia and aecia on lettuce, uredinia and telia on sedges (*Carex* spp.). Eradication of sedges therefore affords a means of control. Endive and chicory are also subject to attack by the lettuce rust fungus.

SCLEROTIUM ROT, caused by *Corticium rolfsii* (page 255), is occasionally recorded on lettuce and endive in the southern USA (10). The fungus is favoured by warm wet conditions, and attacks outer leaves in contact with the soil, giving rise to a silky white mould and spherical resting bodies (sclerotia) (page 84).

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1 BLOK I. & PLAATS-NITERINK A.J. van der (1978) *Pythium uncinulatum* sp. nov. and *P. tracheiphilum* pathogenic to lettuce. *Netherlands Journal of Plant Pathology* **84,** 135–147 illus.

2 ELLIS E.A. (1951) Miscellaneous observations. Lettuce rust. *Transactions of the Norfolk and Norwich Naturalists' Society* **17,** 220.

3 GARIBALDI A. & TESI R. (1971) Resistenza ad *Alternaria porri* f.sp. *cichorii* in *Cichorium* sp. e sua ereditarietà. *Rivista della Ortoflorofrutticoltura Italiana* **55,** 350–355 illus.

4 GOLTZ H. (1958) Kritische Betrachtungen über Rostpilze an Salat anlässlich eines starken Auftretens des Salatrostes (*Puccinia opizii* Bubak). *Nachrichtenblatt für den Deutschen Pflanzenschutzdienst* **12,** 50–55 illus.

5 GRIFFIN M.J. & SIMKIN M.B. (1977) *Mycocentrospora acerina* on outdoor lettuce. *Plant Pathology* **26,** 147–148 illus.

6 MERCIER S., BERTAUX F. & TAMONTE M. (1986) *Mycocentrospora acerina* (Hart.) Deighton, parasite de la laitue de plein air. *Revue Horticole* No. 264, 37–38 illus.

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8 SITEPU D. & BUMBIERIS M. (1981) Stem rot of lettuce caused by a low temperature *Phytophthora porri* in South Australia. *Australasian Plant Pathology* **10**(3), 59–60 illus.

9 TUSET J.J., HINAREJOS C. & GARCÍA J. (1986) *Myrothecium roridum* Tode ex Fries, un tuberculariáceo patógeno de *Euphorbia lathyris* L. y *Lactuca sativa* L. *Investigación Agraria* **1,** 251–258 illus.

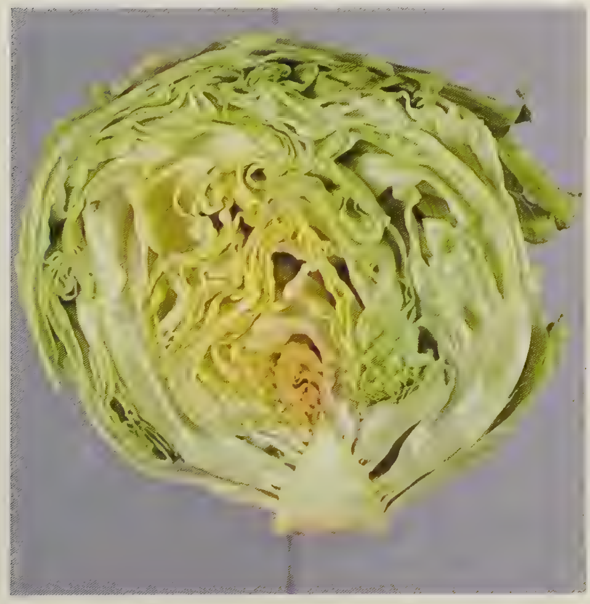
10 WEBER G.F. & FOSTER A.C. (1928) Diseases of lettuce, romaine, escarole, and endive. *Bulletin of Florida Agricultural Experiment Station* No. 195, 303–333 illus.

DISORDERS of lettuce, chicory and endive

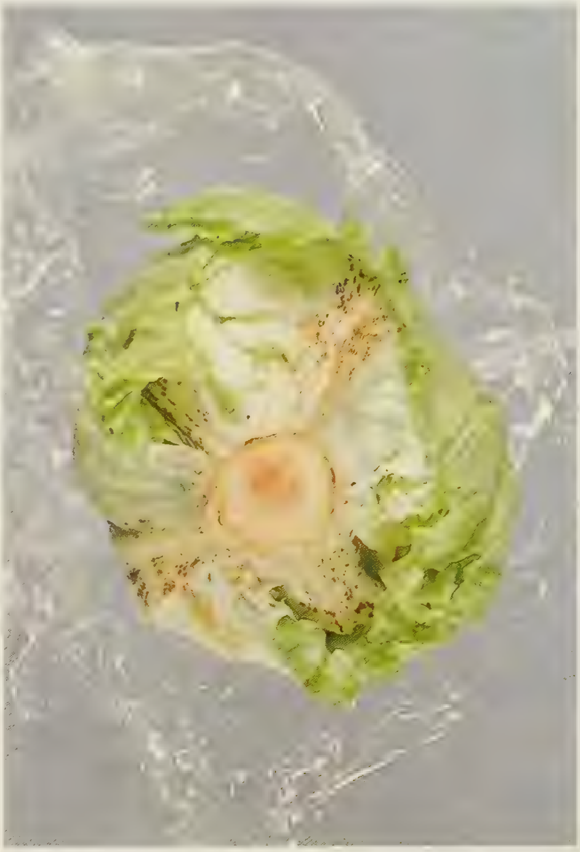
BROWN CORE of chicory is also known as internal browning (3,7). The central part of the 'head', i.e. the stem, appears watersoaked and may be spongy, brown or spotted. Brown core is associated with calcium deficiency, and can be controlled by soaking planting material in calcium chloride (3) or by ensuring adequate fertilization during growth (7).

BROWN RIB of lettuce occurs on the midrib of the outer head leaves, usually at the area of curvature. The causes of this disorder are unknown, but high temperatures during growth seem to favour its development (5). Brown rib differs from pink rib (q.v.) in being generally non-progressive after harvest.

BROWN STAIN of lettuce, a form of carbon dioxide injury, is characterized by slightly sunken oval spots, each possessing a dark brown border (5). The lesions tend to be on or near the midrib, generally towards the base. If the dark borders are absent, brown stain can resemble russet-spotting (q.v.), but may be distinguished from it by frequent association with heart-leaf injury (q.v.), another symptom of carbon dioxide damage. Lettuce cultivars differ in susceptibility to brown stain, but most can be injured by carbon dioxide concentrations of only 1 or 2%, especially during extended shipment or storage. Sensitivity varies not only with cultivar and growing area, but even with the time of day at harvest; in a study in California (2), heads picked in the afternoon were more tolerant of high carbon dioxide levels than were heads picked in the morning.



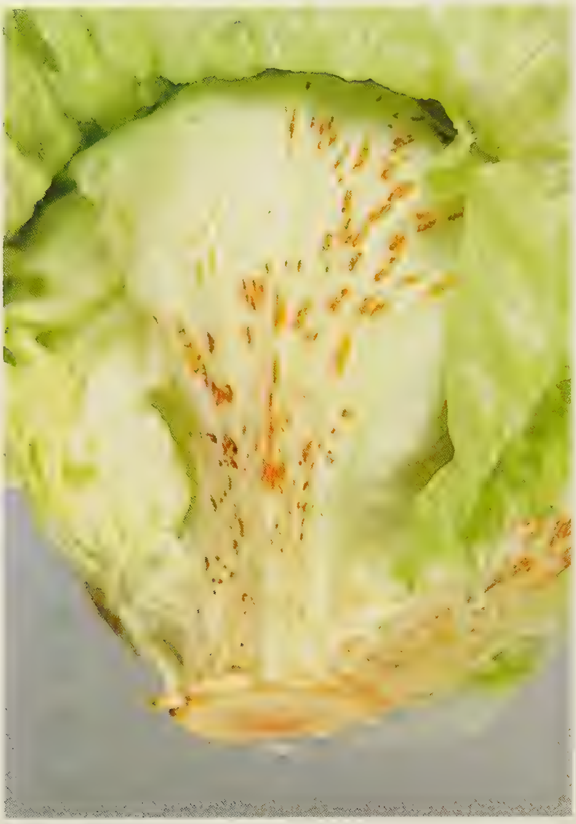
263 Heart-leaf injury of lettuce



264 Russet-spotting of lettuce

GLASSINESS of lettuce is characterized by water-soaked spots, mostly along the leaf margins and bounded by the veins (6). Affected tissue may die, and appearance is marred. This disorder tends to occur under low light intensities such as are experienced during winter in the Netherlands; the plants take up more water than they are able to lose by transpiration (6). Control measures include raising the temperature at night, compacting the soil, and increasing the supply of nitrogen and mineral salts. Cultivars differ widely in their susceptibility to glassiness.

HEART-LEAF INJURY of lettuce, in which the innermost leaves turn brown, is usually the result of an increased concentration of carbon dioxide, though a lack of oxygen may induce similar symptoms.



265 Close-up of russet-spotting

Avoidance of this storage disorder depends on good temperature control to reduce the rate of respiration, and an appropriate air exchange rate to prevent accumulation of respiratory gases (5).

PINK RIB of lettuce affects the midribs of the outer head leaves. A diffuse pink discoloration develops near the base, and although it is at its most intense on the inner (upper) surface, it is often visible externally. The cause is unknown, but symptoms are accentuated by low oxygen levels and above-optimal storage temperatures (5).

RUSSET SPOTTING of lettuce refers to small brown pits, mostly on the midribs but occasionally on the blades (5). Symptoms may develop in heads harvested overmature or stored at too high a temperature, but the primary cause is exposure to ethylene, whether extraneous or from the lettuce itself (9). Prevention of russet spotting is therefore dependent on appropriate harvesting and cooling, and segregation of lettuce from commodities or machinery emitting ethylene. Controlled atmosphere storage is beneficial (4). A post-harvest calcium dip reduces susceptibility to russet spotting but has the disadvantage of reducing crispness in crisphead lettuce. Dipping in certain growth regulators may be preferable.

TIPBURN refers to a browning of the leaf margins and is of major concern to lettuce and chicory growers. It is essentially a field disorder, though the symptoms have been known to intensify after harvest. The fundamental cause is an inadequate distribution of calcium and other minerals within the plant (1,10). Predisposing factors include hot weather and low soil moisture. Some lettuce cultivars show resistance to tipburn (8) but none is immune. Control must therefore depend on careful management of soil moisture and fertility (10).

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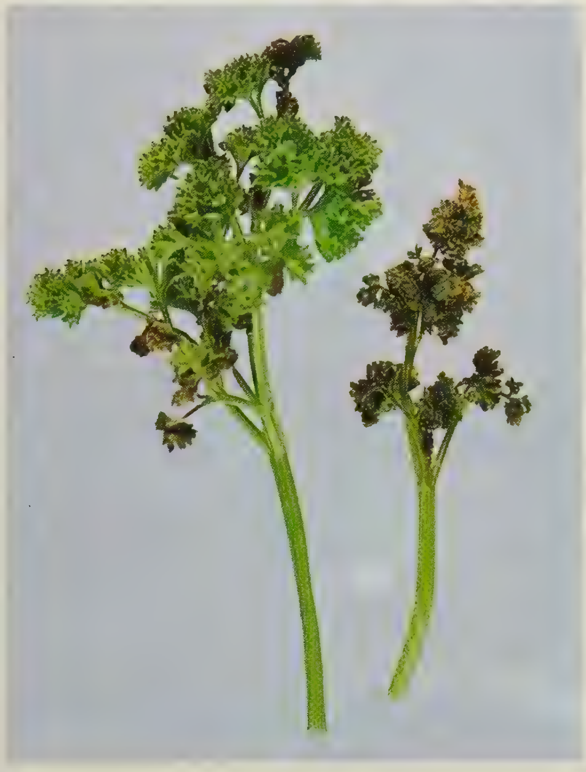
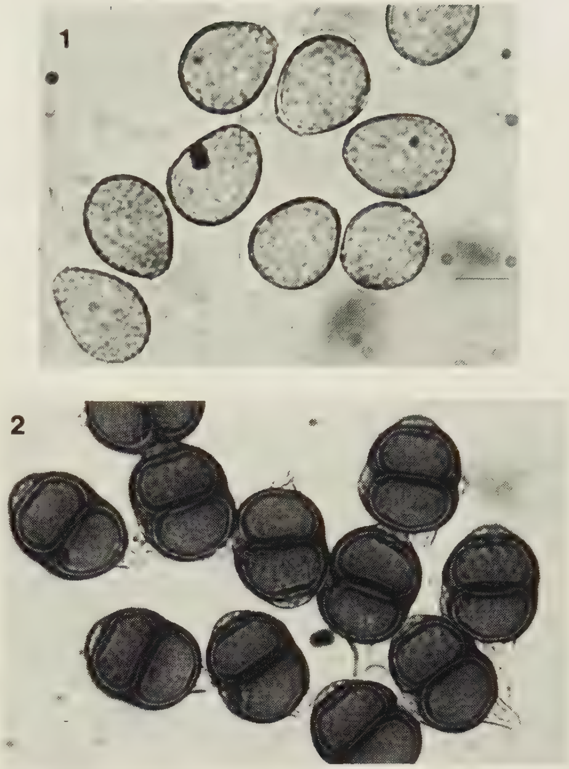
Mint, parsley, cress and watercress

Mint may be any of several species of *Mentha*, belonging to the Labiatae, a family of aromatic herbs native to central Europe. The one most commonly sold fresh is *M. spicata* L. or spearmint, which is especially popular in Britain for making mint sauce to accompany meat. The leaves are prone to serious wilting; unless immediate sale is possible, the freshly harvested sprigs should be packed in perforated plastic film and cooled rapidly to 0°C. The use of 'top ice' is beneficial during transit. High humidity refrigerated storage permits a storage life of up to 4 weeks (4). The most important disease of mint is rust caused by *Puccinia menthae* Pers., of which there are several races, some attacking spearmint and others peppermint. If the small brown pustules are numerous, the mint may be unmarketable. Control measures include field hygiene and the use of healthy planting material.

Parsley, *Petroselinum crispum* L., is a member of the carrot family (Umbelliferae). Its foliage is rich in vitamin C, and it has been used as a garnish and flavouring since the time of the Greeks and Romans. Early varieties possessed finely divided flat leaves, and this type is still common in Mediterranean countries (8). Elsewhere there is a preference for curly-leaved cultivars.



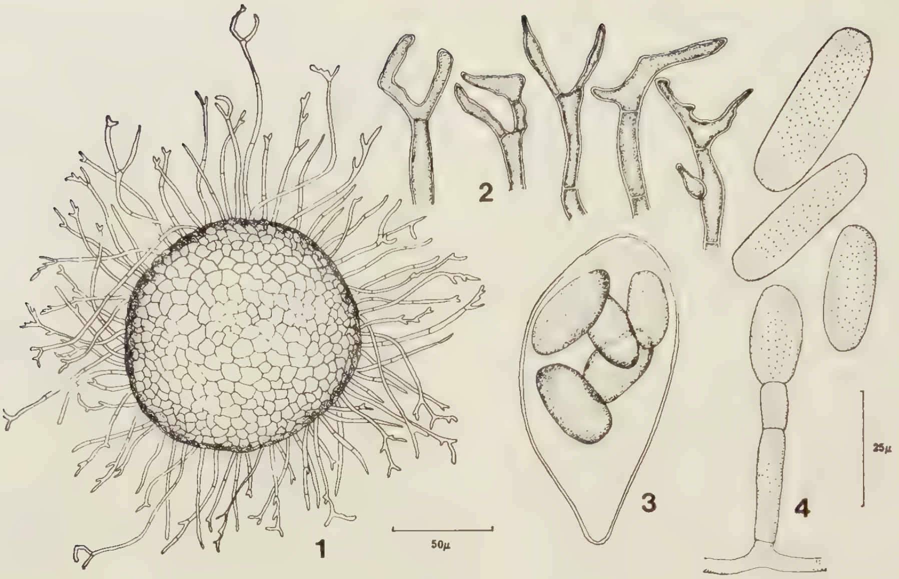
266 Alternaria rot of parsley



267 *Puccinia menthae* (from CMI Descr. No. 7, Laundon & Waterston 1964) 1 urediniospores × 600
2 teliospores × 600

Harvested parsley has a high rate of respiration and, at temperatures above 10°C, the foliage undergoes senescent yellowing within a day or so. Unless intended for immediate local sale, parsley should be promptly cooled to 0°C (1). The other major hazard is wilting, which can be minimised by the use of perforated plastic film packaging. 'Top ice' helps to maintain optimal temperature and humidity during transit. Refrigerated storage for up to 2 months is feasible; controlled atmosphere storage is reported to assist in maintaining green colour (1). For retail sale, unwrapped parsley benefits from immersion of its stems in clean water, to prevent wilting.

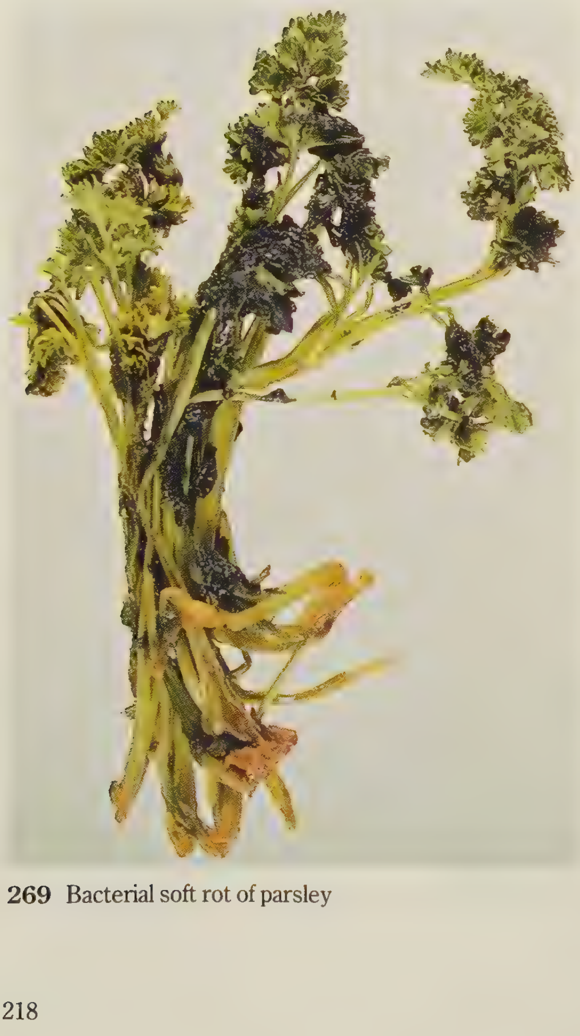
Bacterial soft rot, caused by *Erwinia carotovora* (page 271), is characterised by the development of dark green slime, as the tissue is broken down completely. Decay is favoured by warm moist conditions, and is held in check by refrigeration. Leaf spots may be caused by *Cercospora petroselini* Sacc., *Phyllosticta petroselini* Rothers, *Septoria petroselini* Desmazières, and species of *Alternaria* and *Stemphylium* (5). They are controlled by the use of fungicide sprays. Phytophthora rot may be caused by *Phytophthora*



268 *Erysiphe heraclei* (from CMI Descr. No. 154, Kapoor 1967) 1 cleistothecium 2 branched appendages 3 ascus and ascospores 4 conidia and conidiophore

nicotianae var. *parasitica* (page 76). This disease has been studied in Hawaii (3), and it was established that symptoms develop in the crop only in hot weather (soil temperature of 30°C or above). Powdery mildew is caused by *Erysiphe heraclei* Schleich ex DC., of which several races exist, with differential virulence on carrot, fennel and parsley (8). The fungus can be transmitted in seed. Watery soft rot is caused by *Sclerotinia sclerotiorum* (page 47), which can attack the growing crop. Especially susceptible are closely spaced bushy plants such as those forced under glass (6). Harvested parsley with incipient infection is subject to decay.

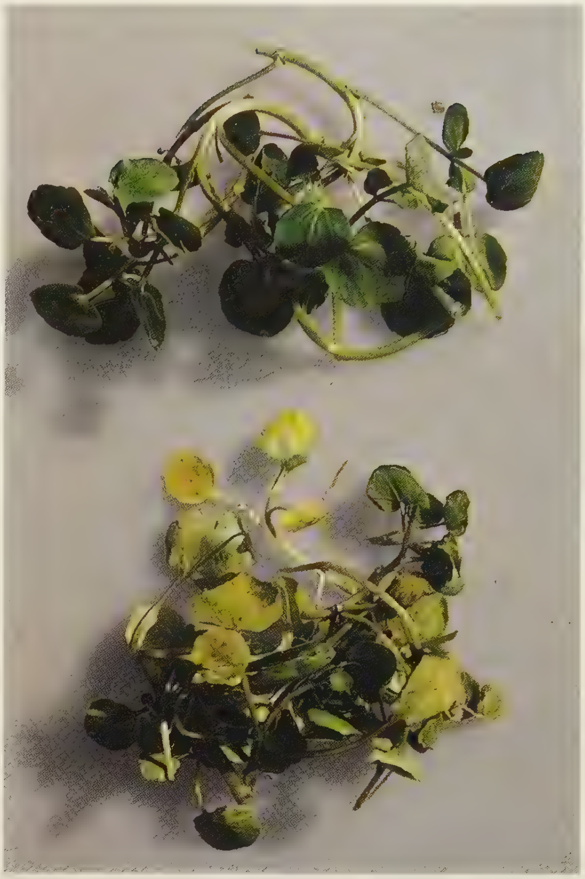
Salad cress describes the seedlings of several members of the family Cruciferae which are used as a salad ingredient and garnish. True cress is *Lepidium sativum* L., which has deeply divided tri-lobed seed-leaves (cotyledons). More common is white mustard, *Brassica alba* (L.) Rabenh., with rounded bi-lobed seed-leaves similar to those of rape, *B. napus* L., which is also used in this way. The seedlings, often a mixture of the three species, are marketed in the peat-filled containers (punnets) in which they were grown, and it is the customer who harvests the crop. The peat needs to be kept moist during marketing, otherwise the seedlings wilt and collapse. A temperature just above 0°C is effective in arresting growth and maintaining a fresh green colour. Even under optimal conditions, however, quality is maintained for no more than a week or so.



269 Bacterial soft rot of parsley

Watercress, *Nasturtium officinale* R.Br., has been known in Europe and Asia for at least two millennia. It is now grown in many temperate and subtropical countries, including the UK (10), France, Germany and the USA (7,9). The plant, a member of the family Cruciferae, is valued for its attractive dark green leaves, which have a pungent taste and are rich in vitamins (9). Watercress grows in running water; it was formerly gathered from the wild, but the crop is now commercially produced in specially constructed 'beds' which require a constant supply of clear alkaline water (7,9). Diseases include leaf spots caused by various fungi, for example *Cercospora nasturtii* Pass. (2).

Harvested watercress is extremely perishable. Unless bunches are placed in water or otherwise protected, the foliage wilts rapidly (9). In warm conditions the lower leaves quickly fade to yellow, and both stems and leaves may become slimy as a result of bacterial soft rot (10). Quality can be maintained only by prompt cooling after harvest (4). Hydro-cooling is advantageous, as moisture loss from the watercress is thereby minimised (10). Optimal holding conditions are a temperature of 0°C and humidity close to saturation; 'top ice' is therefore beneficial (9,10). Watercress is best marketed soon after harvest, though if necessary it may be cold-stored for 2 to 3 weeks (4). Plastic film packaging helps to prevent wilting during marketing (4).



270 Fresh and senescent watercress

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Rhubarb

Rhubarb cultivation has a long history, though originally the crop was grown not for food but as the source of a drug (6). The genus *Rheum* (of the family Polygonaceae) had its origins in eastern Asia, and there are numerous species, all with large coarse leaves arising from a swollen underground stem or rhizome. It was the purgative properties of the dried rhizomes which first brought some species to prominence, and medicinal rhubarbs (*R. palmatum* L. and *R. officinale* Baill.) have been grown in China for thousands of years (6). Dried rhizomes and roots were first exported to Europe during the Middle Ages, and such was the demand for them that eventually, in the 16th century, living rhubarb was brought to Britain. The introduced species was, however, *R. rhabarbarum* (syn. *R. rhaponticum* L.), of scant medicinal value. It was some time later that the English discovered that the succulent leaf stalks were good to eat. Cut into pieces and stewed with sugar, rhubarb became popular in pies and tarts. In Britain it is loosely referred to as a 'fruit' because of its use as a dessert; in the USA, however (to which it was introduced by Italian immigrants), rhubarb is more properly classed as a vegetable in recognition of its botanical structure. Only the stalks are consumed; the leaf blades are poisonous, owing to the presence of oxalates and other compounds (6).

Culinary rhubarb is produced in several temperate countries, for example the UK (1,9), the Netherlands, West Germany, Austria, Australia, New Zealand, Canada and parts of the USA (2,3,4,6,8). It has recently been established in Florida as a winter crop. In the tropics rhubarb grows well only at high altitude (10). This is because a cold spell (temperature below 4°C) is necessary to break dormancy in the rhizome and induce shoot formation. The leaf stalks are green, pink or red, according to cultivar and method of production. Rhubarb grown in the open field is harvested in spring and summer and, in the USA at least, most of the crop goes for processing. Earlier in the year rhubarb can be 'forced' by planting rhizomes (previously held in cold storage) in a dark humid building in which the temperature is maintained at 10° to 12°C. Such conditions encourage the production of tender pink leaf stalks which are much sought after in the fresh market. Rhubarb production can be severely limited by virus diseases, and new techniques are being used to produce healthy planting material (9). Leaf-spotting and stalk-spotting fungi are best controlled by sprays applied early in the season; once the leaves have expanded fully, it becomes difficult to obtain adequate fungicide coverage.

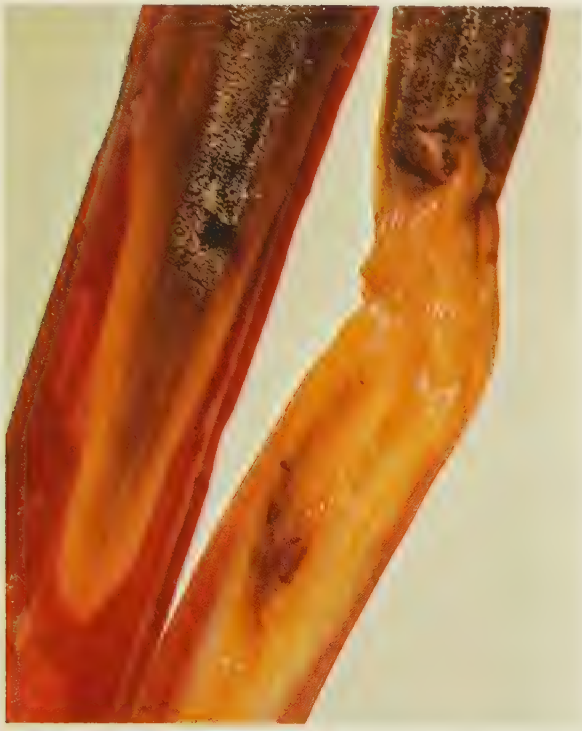
Post-harvest handling practices are directed towards the prevention of wilting (4,10). Some years ago it was customary to market rhubarb with leaf blades intact and with the stalks in water (2). Now, however, it is recommended that the leaf blades be removed, although it is preferable if a few mm of leaf remain so as to keep the stalk from splitting (4,10). Immediate marketing is desirable, unless cooling facilities are available. Hydro-cooling is appropriate, but air-cooling after packing is also feasible, provided that temperature reduction is rapid. As a further means of minimising moisture loss, plastic film packaging may be used, in the form of perforated polyethylene liners for the crates or cartons (4,10). Under optimal conditions (0° to 1°C and high humidity), rhubarb can be stored for 2 to 4 weeks.

ANTHRACNOSE is caused by *Colletotrichum erumpens* Sacc., first recorded in the USA (8). Lesions on leaf stalks are oval, soft and watery, and are characterised by numerous minute black structures (acervuli giving rise to conidia). The fungus probably survives in crop debris, and its spores are disseminated by water and insects. Infection (by direct penetration of intact tissue) is favoured by warm, moist growing conditions. Post-harvest decay can be held in check by refrigeration.

BACTERIAL SOFT ROT, in which affected tissues are soft and slimy, may be caused by *Pseudomonas marginalis* (page 142) or by *Erwinia carotovora* (page 271). (*Erwinia rhapontici* in contrast, causes a firmer, brown rot of the rhizome and leaf stalk bases in the field, CMI Descr. 555.) One outbreak of bacterial soft rot was traced to contaminated pond water (used for watering the crop), coupled with improper growing conditions, especially high humidity and poor ventilation in the forcing house (1). Post-harvest infection, usually via the cut stem-ends, may be linked to inadequate renewal and disinfection of hydro-cooling water. Infection by *Pseudomonas marginalis* is especially serious, since this species can progress even in refrigerated storage.

BLUE MOULD ROT is caused by *Penicillium* spp. and sometimes follows phyllosticta rot (q.v.).

GREY MOULD ROT, caused by *Botrytis cinerea* (page 281), is the most widespread and important disease of rhubarb (6). Lesions are soft and brown, spreading rapidly under warm conditions. Quantities of grey-brown spores are subsequently produced. The disease originates in the field where, in moist conditions, moribund plant parts are colonised by the fungus and act as a source of spores. Vigorously growing leaf stalks are resistant to infection, but are nevertheless likely to be superficially contaminated. After harvest they show increasing susceptibility as



271 Grey mould rot

a result of wounding and moisture loss. Control measures include field hygiene, fungicide sprays, and strict attention to post-harvest handling practices.

PHYLLOSTICTA SPOT, caused by *Phyllosticta straminella* Bres., has been recorded in the USA, Canada and Australia. Lesions tend to be small and numerous, oval to elongated, and reddish-brown in colour (8). Minute black bodies (pycnidia giving rise to conidia) are eventually produced, though these may be obscured by the growth of secondary invaders such as blue mould or grey mould. In the field the most obvious symptom is leaf spotting. Infected stalks should be culled in the pack house.

PHYTOPHTHORA ROT may be caused by *Phytophthora cactorum, P. nicotianae* var. *parasitica* or other species, all of which produce whitish mould growth. Leaf stalks are attacked at the base (3), following infection of the rhizome (crown rot) in the field. Warm wet weather favours disease development, and control measures include improvements in drainage and field hygiene. Diseased rhizomes should be removed and burned.

PYTHIUM ROT may be caused by *Pythium splendens, P. ultimum* or other species. Symptoms and control measures are similar to those described for phytophthora rot (q.v.).

RAMULARIA ROT, caused by *Ramularia rhei* Allescher, has been recorded in the USA, Canada, New Zealand and several European countries. Lesions on leaf stalks are oval, brown and sunken; in a humid atmosphere white spores (conidia) are produced. Since leaf-spotting and consequent stem-infection can render an entire crop worthless, preventive fungicide spraying should begin early in the season (7).

SCLEROTIUM ROT, caused by *Corticium rolfsii* (page 255), has been recorded in the USA and in Australia (5). Infection characteristically occurs at the base of the stalk, which becomes covered with bright white mould and, eventually, small (1 mm) spherical resting bodies (sclerotia). Post-harvest rotting by this fungus is completely controlled by refrigeration.

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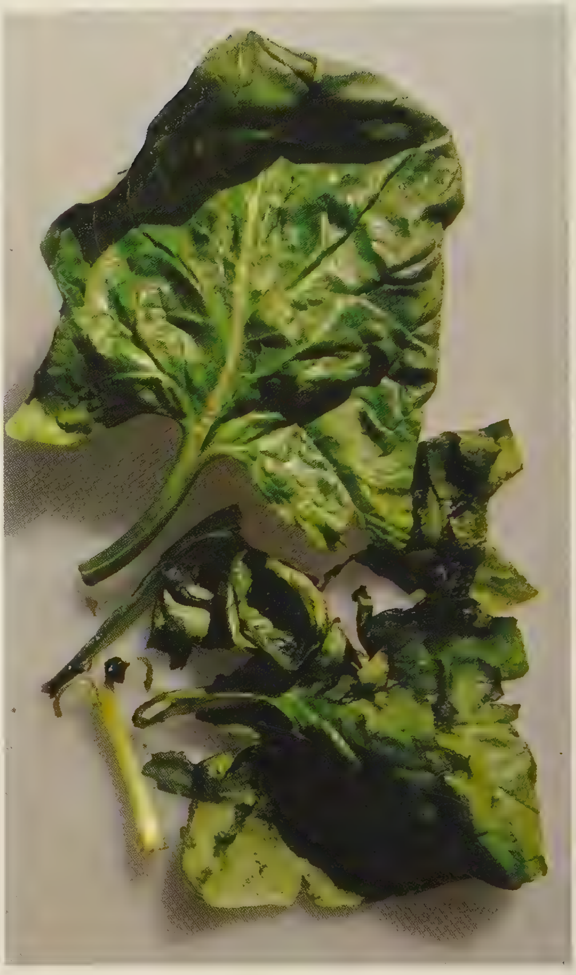
Spinach

Spinach, *Spinacia oleracea* L., is an annual green vegetable grown in cool, moist climates. It is thought to have originated in central Asia, and was introduced into Europe in the Middle Ages. A member of the family Chenopodiaceae (and therefore related to beets, q.v.), spinach is cultivated for its dark green leaves which are rich in vitamins and minerals. The leaves are usually lightly boiled, and can be eaten directly or preserved by freezing or canning. Increasingly spinach is also being used as a fresh salad vegetable. There are three types, according to the form of the leaf, viz. smooth, savoy (crinkled) and semi-savoy. Cultivars with an upright habit can be mechanically harvested.

Because of its high respiration rate, fresh spinach is very perishable and must be promptly cooled if it is not to be sold immediately. Optimal holding conditions are a temperature close to 0°C and a near-saturated atmosphere so as to prevent wilting. Plastic film packaging is appropriate, the usual choice being perforated polyethylene. Controlled atmosphere storage can prolong the life of spinach to about 3 weeks.

There are several other plants which are used in the same way as spinach and whose storage characteristics are similar. Spinach beet is a type of *Beta vulgaris* L. bred for its leaves rather than its root. Swiss chard is *B. vulgaris* var. *cicla,* characterised by broad white leaf stalks which are often served as a separate dish. New Zealand spinach, *Tetragonia expansa* Murray, is in a different family (the Aizoacaceae) and, unlike ordinary spinach, can grow under warm conditions. Tannia spinach, from various species of *Xanthosoma* (q.v.), is popular in the tropics.



272 Bacterial soft rot

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DISEASES of spinach

ANTHRACNOSE may be caused by either of two species of *Colletotrichum*, both confined to spinach. Leaf symptoms include small watersoaked spots which enlarge to become irregular pale patches. Elongate lesions may be found on leaf stalks. Microscopic examination is required to distinguish between *C. spinaciae* Ell. & Halsted (with curved conidia) and *C. spinacicola* (Ell. & Everh.) Sherf & MacNab (with straight conidia). The former can only attack plants which have been weakened by unfavourable growing conditions, while the latter is capable of infecting healthy plants. Both fungi are seed-borne, and may also persist in crop debris. Dissemination is by rainsplash, irrigation water, and the movement of people through the crop. Control measures include crop rotation, seed treatment, improved drainage, and the use of fungicide sprays.

BACTERIAL SOFT ROT may be caused by species of *Erwinia* and *Pseudomonas*. The dark slimy breakdown is accompanied by a disagreeable odour. Predisposing factors include injury to the leaves and storage under warm, moist conditions.

DOWNY MILDEW is caused by *Peronospora farinosa* (Fr.) Fr. f. sp. *spinaciae* Byford. Harvested spinach leaves may show yellowish blotches on the upper surface, with corresponding greyish mould spots on the underside. A humid atmosphere encourages the development of asexual spores (sporangia/conidia). Wet growing conditions can result in entire crop loss in the field. The fungus survives as thick-walled oospores in soil and seed. Control measures have included seed treatment, crop rotation, segregation of spring and winter spinach crops, and the use of fungicide sprays. A more convenient strategy is the use of resistant varieties; some spinach cultivars are completely immune to the disease, though there remains the possibility that new races of the fungus may evolve.

RUST is caused by *Puccinia aristidae* Tracy, and is characterised by pale yellow-green spots which enlarge to form orange pustules. The fungus (which also attacks beets) exists only in areas supporting the growth of its alternate host, salt grass (*Distichlis spicata* (L.) Greene). Ultimate control is therefore by eradication of salt grass; an interim measure is to protect the spinach crop with fungicide sprays.

WHITE BLISTER is caused by *Albugo occidentalis* Wilson, which is related to downy mildew. The alternative epithet 'white rust' is therefore inappropriate, since the fungus is not a true rust. The white blister-like pustules do, however, resemble 'rust-cups' in shape; they tend to be confined to the undersurface of the leaf, and may also develop on leaf stalks. The pustules themselves remain small (2 to 3 mm), but the surrounding tissue loses its green colour and eventually turns brown.

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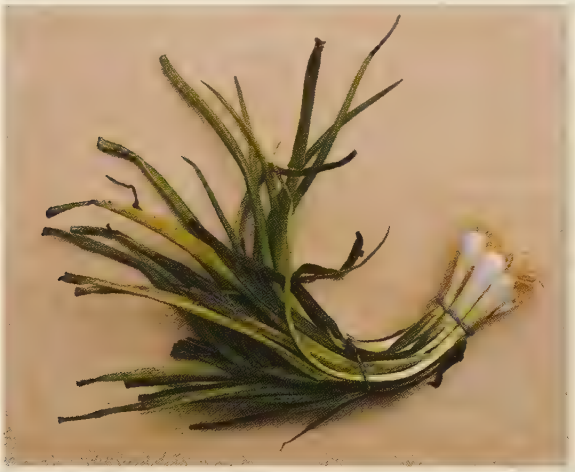
Spring onions, leeks and chives

Spring onions, also known as green onions, salad onions or scallions, are immature forms of certain onion cultivars, *Allium cepa* L. (q.v.). They are eaten raw and are appreciated for their pungent taste. The plants are grown from seed, and are harvested while the leaves are still fresh and the bulbous base is still small (6). Being immature, they have a high rate of respiration and deteriorate rapidly under warm conditions. The shoots continue to elongate and, if packed horizontally, they curve upwards (6). Prompt cooling to 0°C prevents elongation and helps to minimise wilting and decay. High humidity cold storage permits a holding period of about 1 month. Storage can be extended to 2 months by the use of perforated plastic film packaging and an appropriate controlled atmosphere (6).

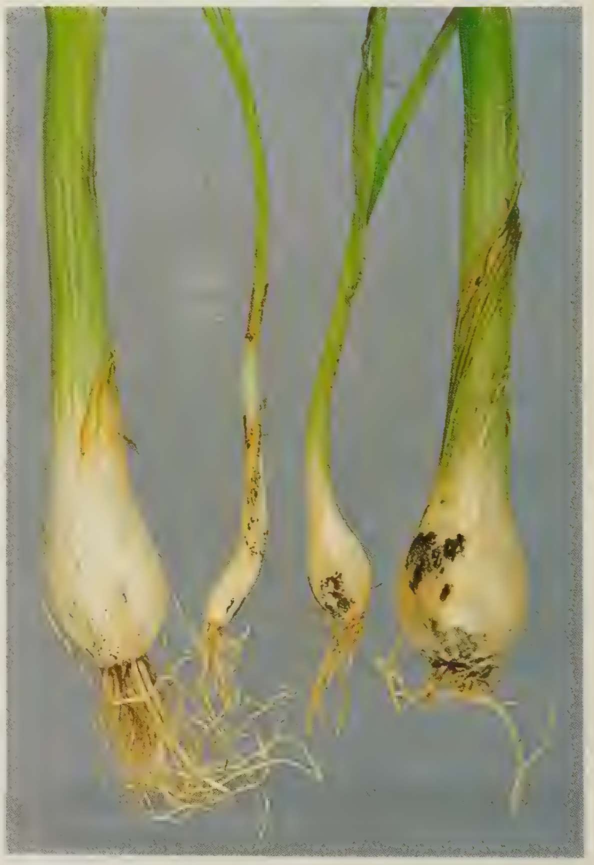


273 Bending and bacterial soft rot of spring onions

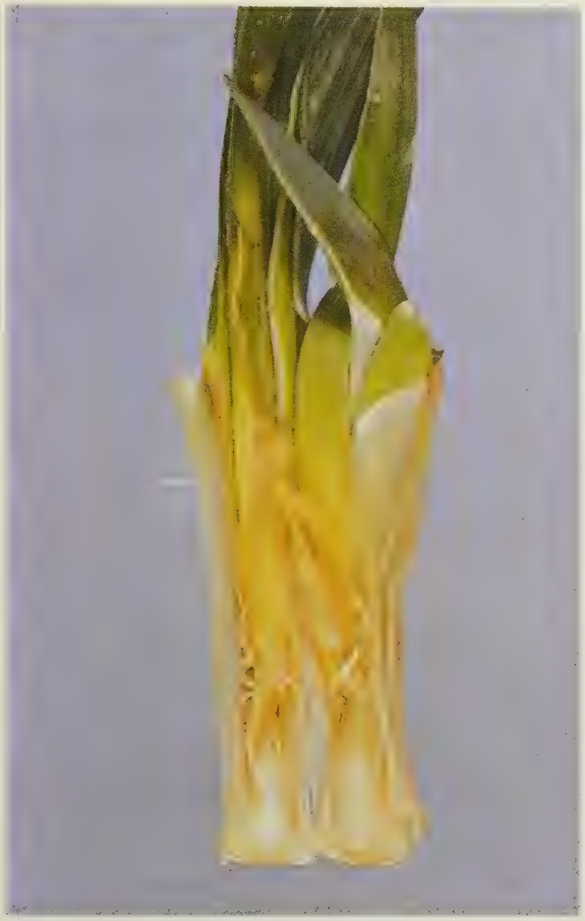
The Japanese bunching onion, *Allium fistulosum* L., has been the principal garden onion in China and Japan since ancient times, but is uncommon in the west. It develops only slightly swollen leaf bases. The young plant may be eaten raw, as a spring onion, or allowed to develop further and used in the same way as a leek (q.v.).

The **leek**, *Allium ampeloprasum* L. var. *porrum* (L.) Gay, is of ancient cultivation in the Levant, and is now of particular importance in northern Europe, for example in France (1,4), the Netherlands, the UK (2) and the Nordic countries (5,9,10). Other producer countries include the USA (7) and Canada (3).

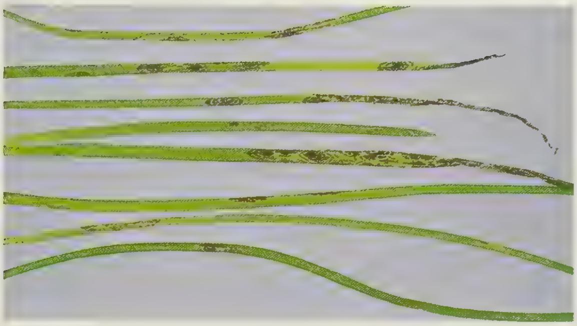
In contrast to spring onions, leeks are normally cooked. The edible portion consists of the elongated bases of the foliage leaves (a false stem), the lower parts of which are white, having been blanched through being underground. At harvest, leeks are trimmed so that only a portion of the green top remains (7). If they are to be stored, leeks should be cooled immediately; they can be held at 0°C and high humidity for 1 to 3 months (7). The use of a controlled atmosphere can extend the storage period to 4 or 5 months (5), though with some loss of quality. Controlled atmosphere storage at −1°C (just above the freezing point of leeks) is optimal but should only be used if accurate temperature control can be guaranteed. Potential storage life decreases substantially with increase in temperature; at 5°C it is less than half that at 0°C (7). The major hazards are wilting, elongation, senescent yellowing, and decay (10). For maintenance of quality, plastic film packaging is desirable, whether used as a liner in wooden storage crates or as a consumer pre-pack. Sealed polyethylene bags have been found to be superior to perforated bags, probably because of the resultant modified atmosphere; no off-odours were noted (7). As well as retarding physiological deterioration, an increased carbon dioxide concentration can inhibit disease development (5). However, some fungal lesions may already be evident on harvested leeks, for example those of rust (q.v.) on the green leaves and white rot (q.v.) of the base (2,4).



274 Smudge of spring onions



275 Bacterial soft rot of leek



276 Chive rust

Chives, *Allium schoenoprasum* L., are native to the northern regions of Europe, Asia and America, but have also been taken to the Antipodes (8). The plant grows in dense clumps; it does not produce bulbs, and is grown for its fine dark green tubular leaves which provide a mild-flavoured garnish. Freshly harvested chives can be kept for 1 to 2 weeks at 0°C and high humidity. Plastic film packaging serves to minimise wilting. A substantial proportion of the crop goes directly for processing.

Chinese chives, *Allium tuberosum* Rottl. ex Spreng., are widely grown in eastern Asia, for example Taiwan, the Chinese mainland, Thailand, the Philippines and Japan. The plant produces rhizomes rather than bulbs, but it is the thin flattish leaves and young flower stalks that are eaten; being strongly flavoured, they are used as a seasoning. The harvested product has a high rate of respiration, and a means of cooling (such as top ice) is desirable during transport to market.

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1 BONNET B. (1976) The leek (*Allium porrum* L.): botanical and agronomic aspects. *Saussurea* **7,** 121–155.

2 BURCHILL R.T., EVERETT B.A., MAUDE R.B. & PEACH L. (1987) Diseases of leeks – biology, resistance and control. *37th Annual Report (1986/87) of the National Vegetable Research Station, Wellesbourne, UK,* pp. 62–63.

3 GARIÉPY Y., RAGHAVAN G.S.V., PLASSE R., THÉRIAULT R. & PHAN C.T. (1985) Long-term storage of cabbage, celery, and leeks under controlled atmosphere. *Acta Horticulturae* No. 157, 193–201.

4 GRILL D. (1985) Les maladies et ravageurs du poireau. *Revue Horticole* No. 262, 39–43 illus.

5 HOFTUN H. (1978) [Storage of leeks. 3. Storage in controlled atmospheres.] *Meldinger fra Norges Landbrukshøgskole* 57, Report No. 75, 46 pp.

6 HRUSCHKA H.W. (1974) Storage and shelf life of packaged green onions. *Marketing Research Report of the United States Department of Agriculture* No. 1015, 21 pp illus.

7 HRUSCHKA H.W. (1978) Storage and shelf life of packaged leeks. *Marketing Research Report of the United States Department of Agriculture* No. 1084, 19 pp illus.

8 KENNELLY A.G. (1963) Growing onions, shallots, chives, and garlic. *New Zealand Journal of Agriculture* **107,** 109, 112–113, 116 illus.

9 KURKI L. (1979) Leek quality changes in CA storage. *Acta Horticulturae* No. 93, 85–90.

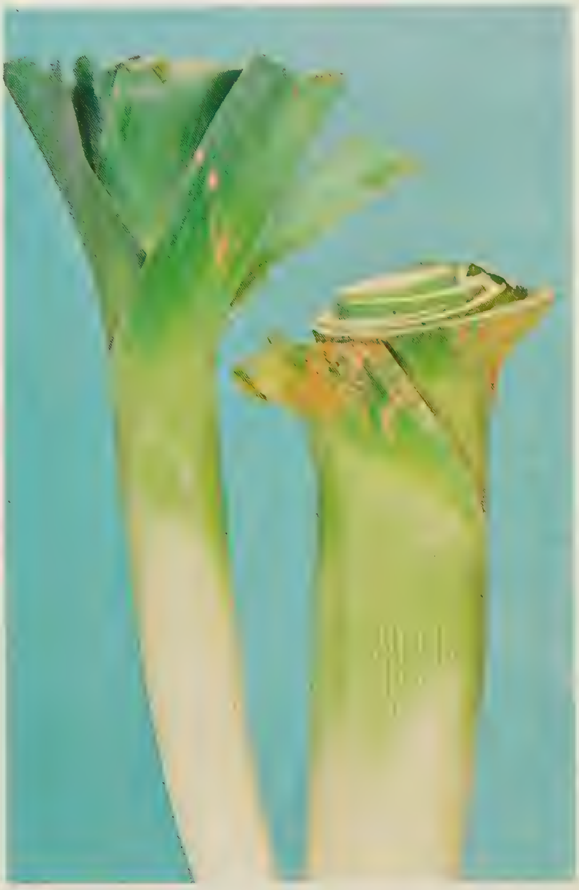
10 SUHONEN I. (1970) On the storage life of the leek. *Acta Agriculturae Scandinavica* **20,** 25–32.

See also:

EMBRECHTS A.J.M. (1988) Onderzoek op Sprenger Instituut leert: prei beter bewaarbaar in CA-cel. *Groenten en Fruit* **43**(27), 52–53 illus.

RUST of spring onions, leeks and chives caused by
Puccinia allii Rud.

277



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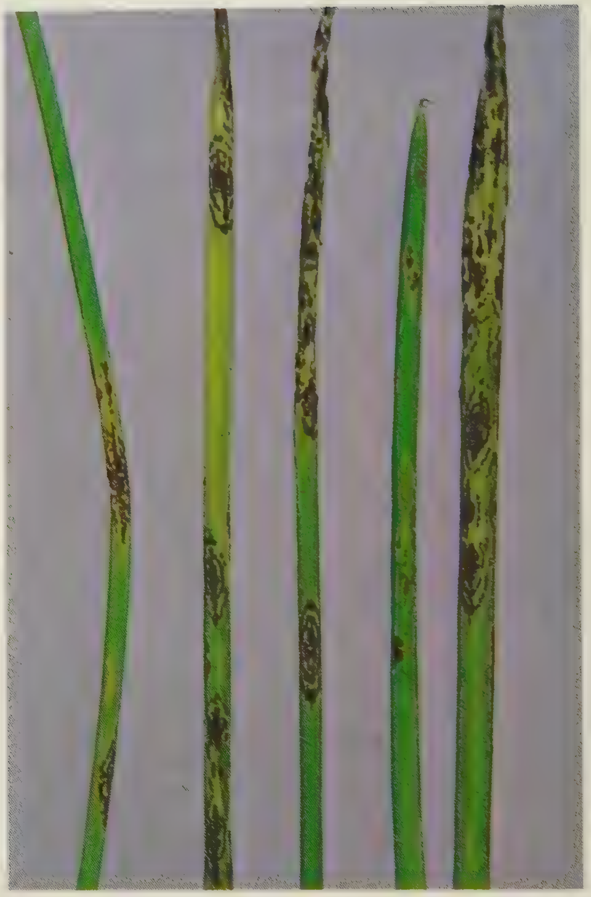
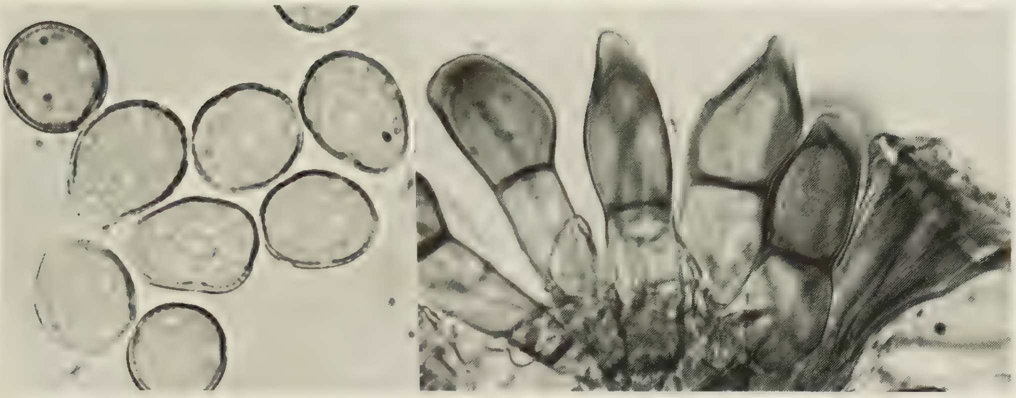


OCCURRENCE Rust of *Allium* species probably occurs in all countries where these crops are grown. Records include onions in Italy (8) and Japan, Chinese chives in Hong Kong (7,9) and leeks in the UK, where the disease is of major importance (1,2,3,4,5, 6,10). A related fungus, *P. mixta* Fuckel, occurs on chives in the UK (5).

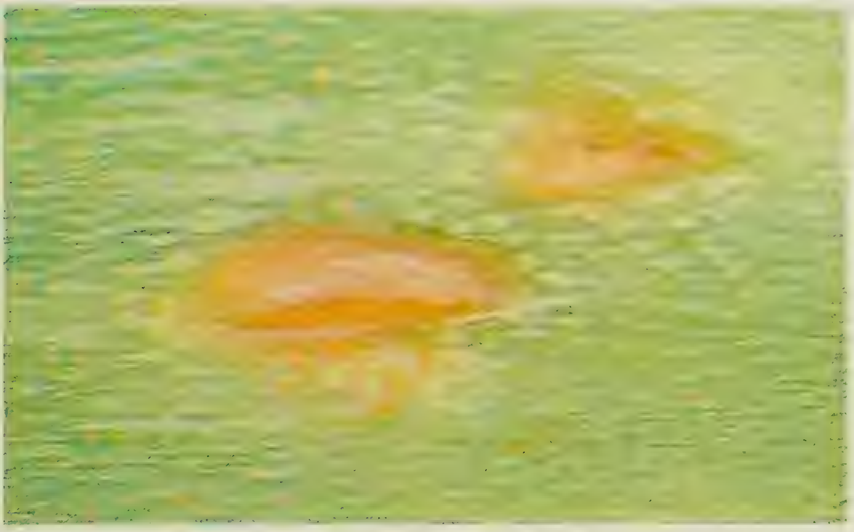
SYMPTOMS It is the leaves and stem of the plant which are attacked, hence the significance of the disease on harvested spring onions, leeks and chives. Occasionally there is no progression beyond the formation of pale spots. In susceptible cultivars of leek, however, the symptoms are bright orange circular to elongate pustules, mostly 1 to 3 mm in length, between the veins of the leaf (2). Masses of urediniospores are released. In spring onions and chives, the orange pustules are succeeded by dark brown pustules containing teliospores.

BIOLOGY The fungus has an elaborate life cycle. The teliospores give rise to basidiospores which are dispersed in the air and may infect other hosts, leading to the formation of spermatia and aeciospores. Allium species are infected when they are under stress, for example in soils which are too dry or too wet, or when the foliage is too dense after excessive use of fertiliser (2). Rust development is inhibited by temperatures below 10°C (1) and especially by frost (6).

CONTROL Control measures include the use of resistant varieties, crop rotation, improvement of drainage, careful choice of fertiliser, and the application of fungicide sprays. There are several reports of successful pre-harvest spraying (6,7), and a recommendation that in addition any plants showing symptoms should be removed and burned (8). There have been laboratory investigations into the possibility of using biological control methods (3,10), and field trials using crop mixtures in which individual cultivars have differing degrees of resistance to the disease.



279 *Puccinia allii* (from CMI Descr. No. 52, Laundon & Waterston 1965) Urediniospores, teliospores and paraphyses × 600



280 Uredinia on leek leaf

1 BURCHILL R.T. & EVERETT B.A. (1985) Leek rust – epidemiology. *35th Annual Report (1984) of the National Vegetable Research Station, Wellesbourne, UK,* pp 71–73.

2 DIXON G.R. (1976) Observations on the incidence of leek rust in NIAB trials. *Journal of the National Institute of Agricultural Botany* **14,** 100–104.

3 DOHERTY M.A. & PREECE T.F. (1978) *Bacillus cereus* prevents germination of uredospores of *Puccinia allii* and the development of rust disease of leek, *Allium porrum,* in controlled environments. *Physiological Plant Pathology* **12,** 123–132.

4 HARRISON J.M. (1987) Observations on the occurrence of telia of *Puccinia porri* on leeks in the UK. *Plant Pathology* **36,** 114–115 illus.

5 JENNINGS D.M., FORD-LLOYD B.V. & BUTLER G.M. (1990) Morphological analysis of spores from different *Allium* rust populations. *Mycological Research* **94,** 83–93 illus.

6 JONES O.W. (1985) Comparison of fungicides for control of rust on leeks, 1982–83. *Tests of Agrochemicals and Cultivars* No. 6 *(Annals of Applied Biology* **106,** Supplement), 54–55.

7 LEATHER R.I. (1968) A note on the control of rust (*Puccinia allii* Rud.) on Chinese chives in Hong Kong. *Agricultural Science, Hong Kong* **1,** 13–16.

8 MARCHETTI L. (1977) La ruggine della cipolla. *Informatore Fitopatologico* **27**(1), 33.

9 SO M.L., TSE K., POON O.S., WONG C.F. & LEE M.C. (1988) Viability and germinability studies on uredospores of *Puccinia allii. Korean Journal of Plant Pathology* **4,** 197–201.

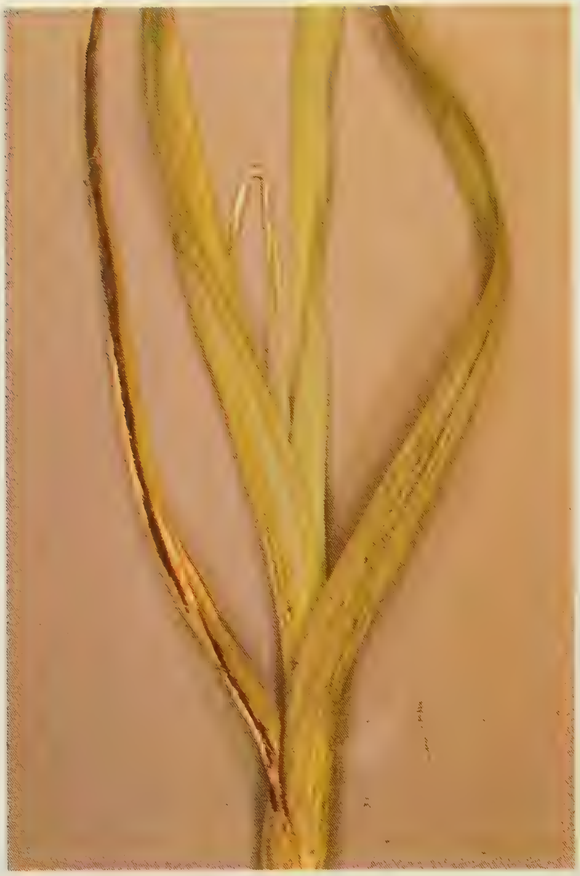
10 UMA N.U. & TAYLOR G.S. (1987) Parasitism of leek rust urediniospores by four fungi. *Transactions of the British Mycological Society* **88,** 335–340.

SMUT of spring onions, leeks and chives caused by

Urocystis cepulae **Frost**

(syn. *Urocystis magica* Pass. ap. Thüm)

281



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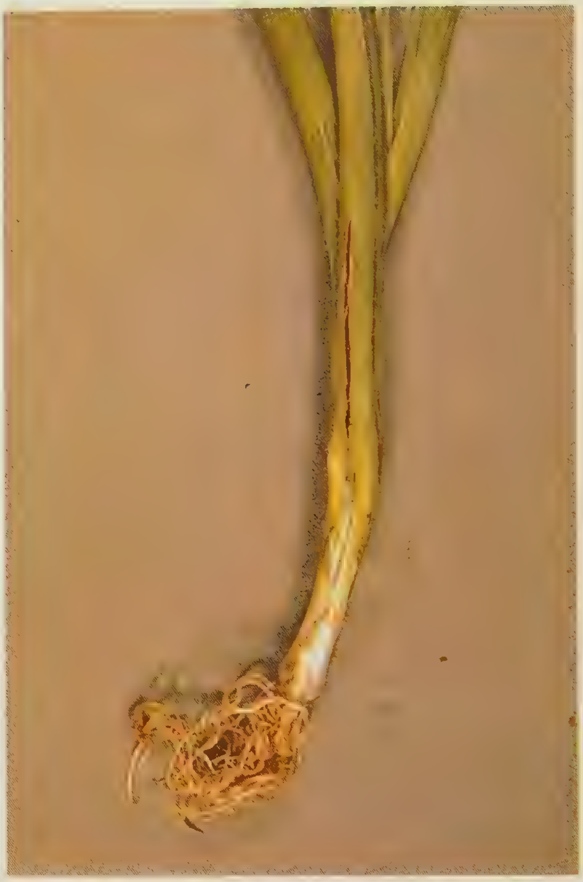

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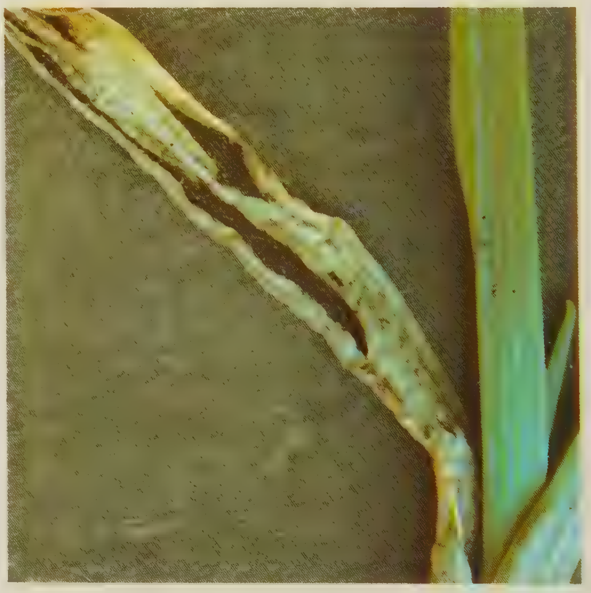
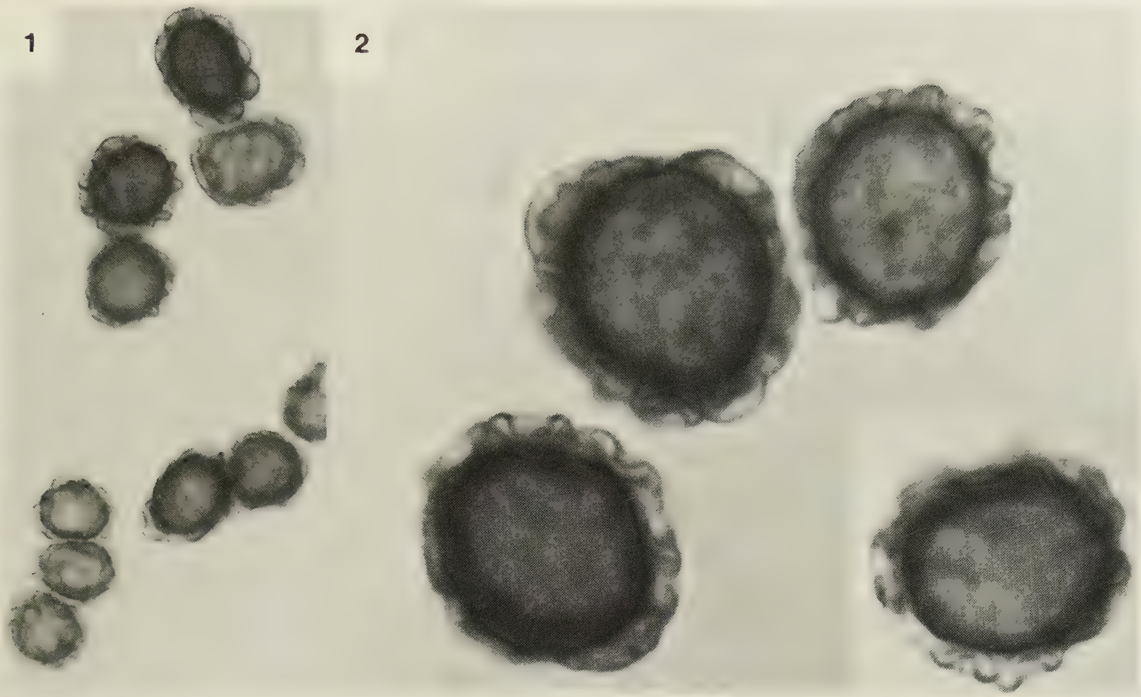


OCCURRENCE Smut can cause heavy losses in temperate growing areas such as the UK (9), Poland (5), Canada (2,8), the USA (10) and New Zealand (4). There are also reports from Egypt (3) and India (7).

SYMPTOMS Elongated brown to black pustules develop on the surface and within the tissues, and dark spore masses are eventually released. Smutted onion bulbs undergo shrinkage and show an increased susceptibility to attack by secondary organisms (1).

BIOLOGY Smut spores (ustilospores) can survive for years in the soil (6), but infection occurs only when the plants are very young (up to 3 weeks after germination) and if the temperature is not too high, the optimal range for infection being 15° to 20°C (5,10). Severely infected seedlings are killed, but slightly infected plants, though stunted, may survive to maturity.



284 *Urocystis cepulae* (from CMI Descr. No. 298, Mulder & Holliday 1971) 1 ustilospores × 800 2 ustilospores × 2000

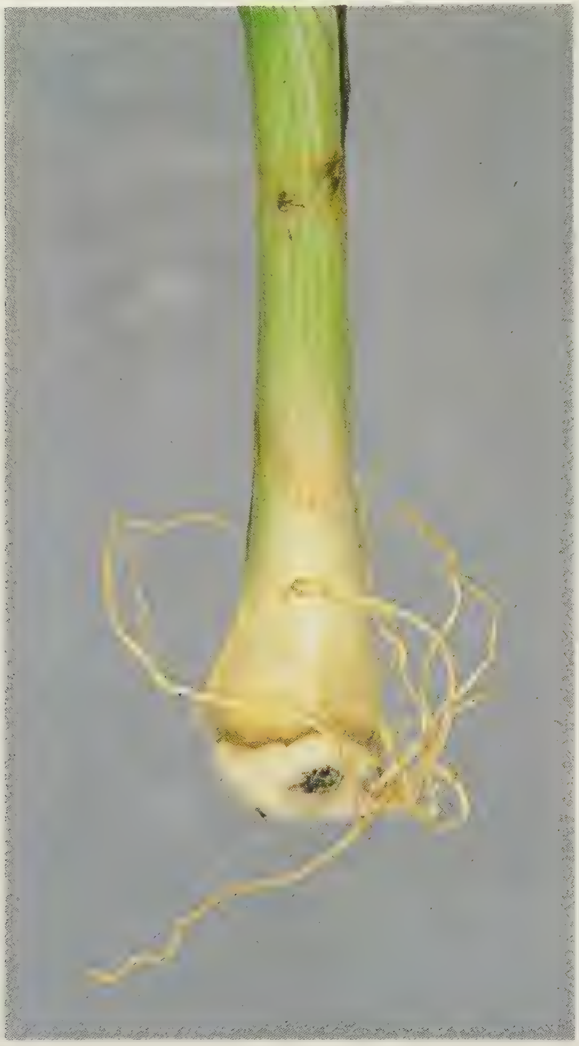
CONTROL Seed may be protected by application of fungicide either before or during planting (2,7,9). Neither seed-treatment nor soil-treatment is entirely satisfactory, and an alternative strategy is to sow the crop later in the season when temperatures are high enough to inhibit infection and disease development (9).

---

1 ANDERSON P.J. & OSMAN A.V. (1924) The smut disease of onions. *Bulletin of Massachusetts Agricultural Experiment Station* No. 221, 29 pp illus.
2 CREVIER H. & STEWART K.A. (1988) Effect of a systemic fungicide gel mixture on onion growth and smut. *Canadian Journal of Plant Science* **68,** 557–559.
3 EL-SHEHABY A.I. & MOHAMED H.A. (1985) Some side effects of fungicides on onion plants. *Agricultural Research Review, Cairo* **63**(2), 89–98.
4 HARROW K.M. (1970) Tests show MeBr fumigation suitable for eradication of onion smut. *New Zealand Commercial Grower* **25,** 27.
5 KOCHMAN J. & MACIAS W. (1974) Effect of soil type, pH, moisture and temperature on the phytotoxicity and effectiveness of fungicides of the PCNB and thiram groups against onion smut (*Urocystis magica* Pass.). *Acta Agrobotanica* **27,** 85–103.
6 TACHIBANA H. & DURAN R. (1966) Comparative survival of teliospores and mycelia of the onion smut fungus in soil. *Phytopathology* **56,** 136–137.
7 URS N.V.R., GOVINDU H.C. & MEHTA P.R. (1963) Present status of onion smut in India and its control. *Current Science* **32,** 445–447.
8 UTKHEDE R.S. & RAHE J.E. (1980) Screening world onion germplasm collection and commercial cultivars for resistance to smut. *Canadian Journal of Plant Science* **60,** 157–161.
9 WALE S.J. & GOATS G.C. (1984) Effect of cultivar, date of sowing and seed treatment on the incidence of smut (*Urocystis cepulae*) in leek. In *Crop Protection in Northern Britain 1984,* pp. 412–416. Dundee, UK: Scottish Crop Research Institute.
10 WALKER J.C. & WELLMAN F.L. (1926) Relation of temperature to spore germination and growth of *Urocystis cepulae. Journal of Agricultural Research* **32,** 133–146 illus.

WHITE ROT of spring onions, leeks and chives caused by ***Sclerotium cepivorum*** **Berk.**

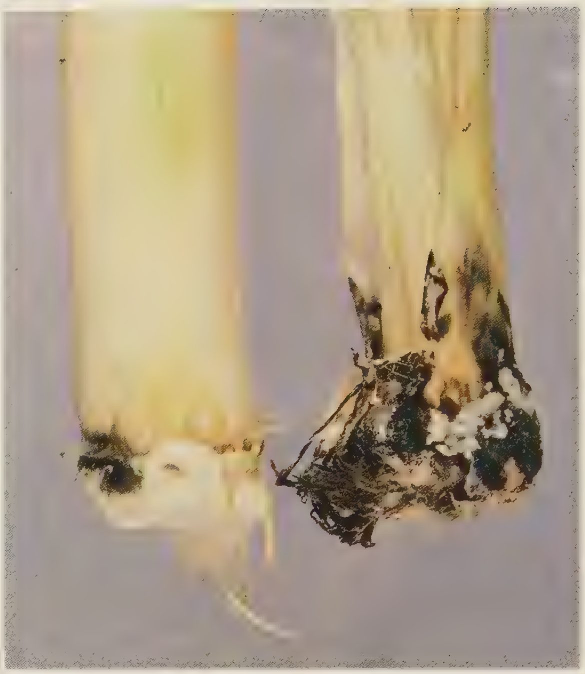
285



OCCURRENCE White rot is becoming increasingly prevalent and can cause heavy losses, chiefly in temperate growing areas such as the UK (1,2,5,9), New Zealand (6), Canada (2,10) and the USA (4), but also in warmer countries such as Egypt (7) and Brazil (8). It is of particular importance on spring onions, but also affects leeks, chives, bulb onions and garlic (3,4,8).

SYMPTOMS The disease is manifest as a soft decay and the development of fluffy white mould at the base of the bulb. The diagnostic symptom is the formation of minute hard black spherical bodies (sclerotia), 0.2 to 0.5 mm in diameter, which develop on the surface and within the fleshy scales (10). Occasionally much larger sclerotia are formed as well. Eventually the bulb rots completely.

286



BIOLOGY Sclerotia persist in the soil for many years (9), and can survive passage through the digestive tract of sheep and goats grazing on crop debris (7); thus the fungus can be transmitted to new areas. Stimulated by volatiles produced by *Allium* species (3), the sclerotia germinate to form mould strands which invade the roots of the growing crop (9). At favourable temperatures (15° to 20°C) severe losses may occur in the field; in addition, recently infected plants may be harvested in apparently good condition, only to decay during storage and marketing.

CONTROL A range of measures has been investigated, and integrated control is desirable (2,3). Flooding the soil has the effect of reducing numbers of viable sclerotia (2). Solarization (page 132) is

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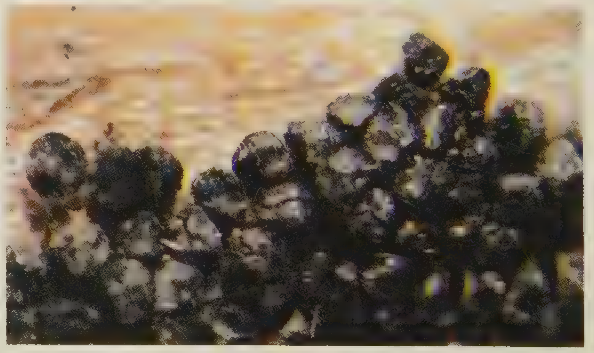

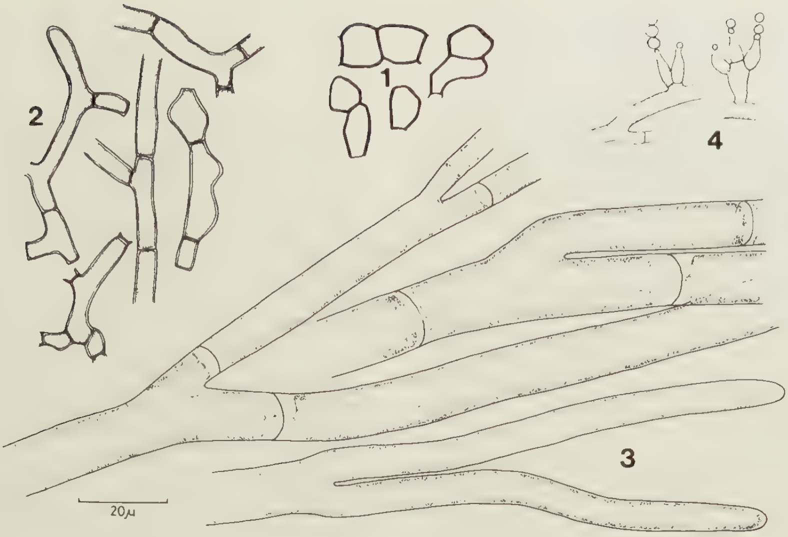

288 *Sclerotium cepivorum* (from CMI Descr. No. 512, Mordue 1976) 1 cells from sclerotium rind 2 cells from sclerotium medulla 3 hyphae 4 spermatia

feasible in countries with a hot dry season (3). Chemical treatments include applications of fungicide to the seed or sets, to the growing plants and to the harvested crop, though such methods are not always effective (3,4,5,8,10). Efforts have been made to disinfest soil by introducing a specific fungus or bacterium which attacks and destroys sclerotia (1,6). Alternatively the sclerotia can be stimulated to germinate in the absence of the host crop by the addition of onion or garlic extracts to the soil (3); by the time the crop is planted, most of the sclerotia have decayed. Work is in progress to increase the efficacy of these control measures (3), and also to breed new cultivars with enhanced disease resistance (10).

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1 AHMED A.H.M. & TRIBE H.T. (1977) Biological control of white rot of onion (*Sclerotium cepivorum*) by *Coniothyrium minitans. Plant Pathology* **26,** 75–78.
2 BANKS E. & EDGINGTON L.V. (1989) Effect of integrated control practices on the onion white rot pathogen in organic soil. *Canadian Journal of Plant Pathology* **11,** 268–272.
3 COLEY-SMITH J.R. (1990) White rot disease of *Allium*: problems of soil-borne diseases in microcosm. *Plant Pathology* **39,** 214–222.
4 CROWE F.J., HALL D.H., GREATHEAD A.S. & BAGHOTT K.G. (1980) Inoculum density of *Sclerotium cepivorum* and the incidence of white rot of onion and garlic. *Phytopathology* **70,** 64–69.
5 ENTWISTLE A.R. (1986) Evaluation of SC9906 for the control of white rot in leeks. *Tests of Agrochemicals and Cultivars* No. 7 (*Annals of Applied Biology* **108,** Supplement), 54–55.
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8 RESENDE M.L.V. de, ZAMBOLIM L. & CRUZ FILHO J. da (1987) Eficiência de fungicidas no controle da podridão branca do alho (*Allium sativum*), de acordo com o nível de escleródios de *Sclerotium cepivorum* no solo. *Fitopatologia Brasileira* **12,** 71–78.
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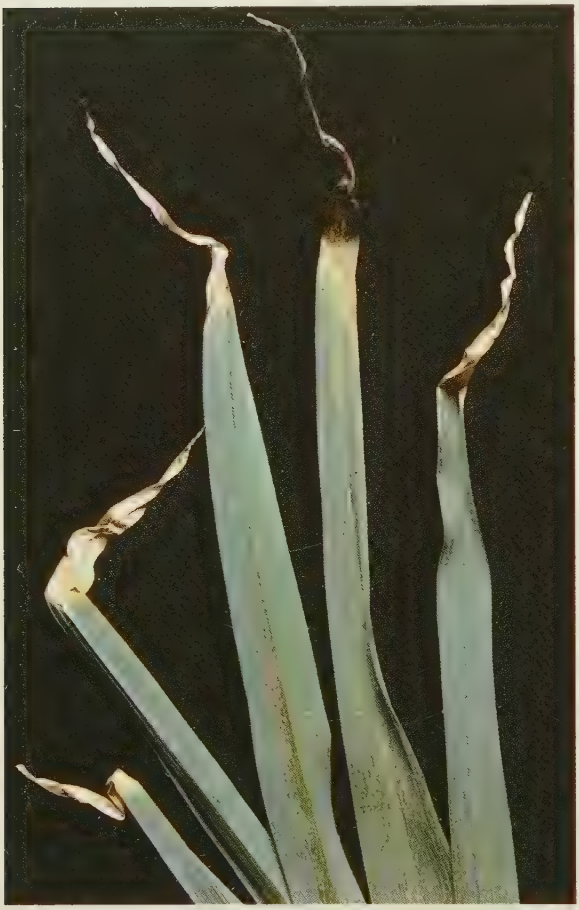
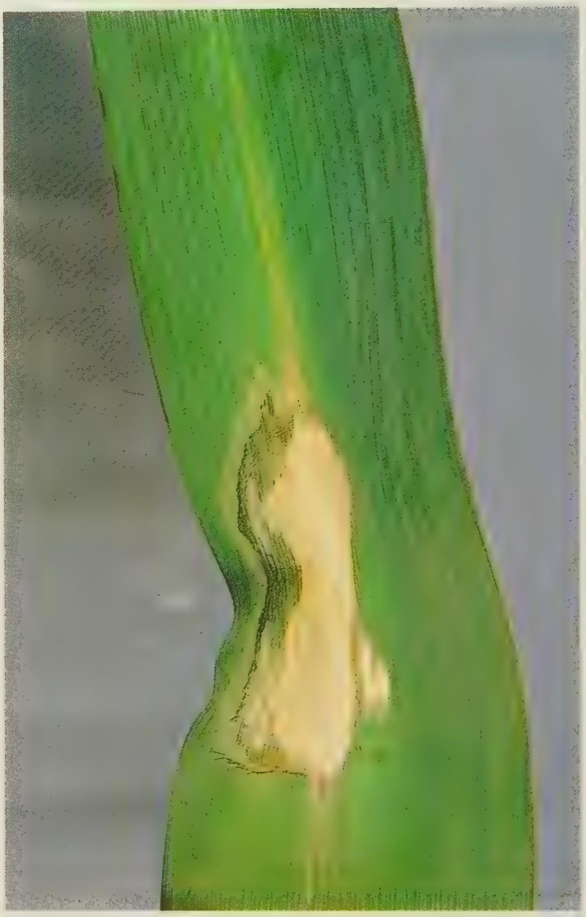
WHITE TIP of spring onions and leeks caused by *Phytophthora porri* Foister



289 Tip lesions on leek



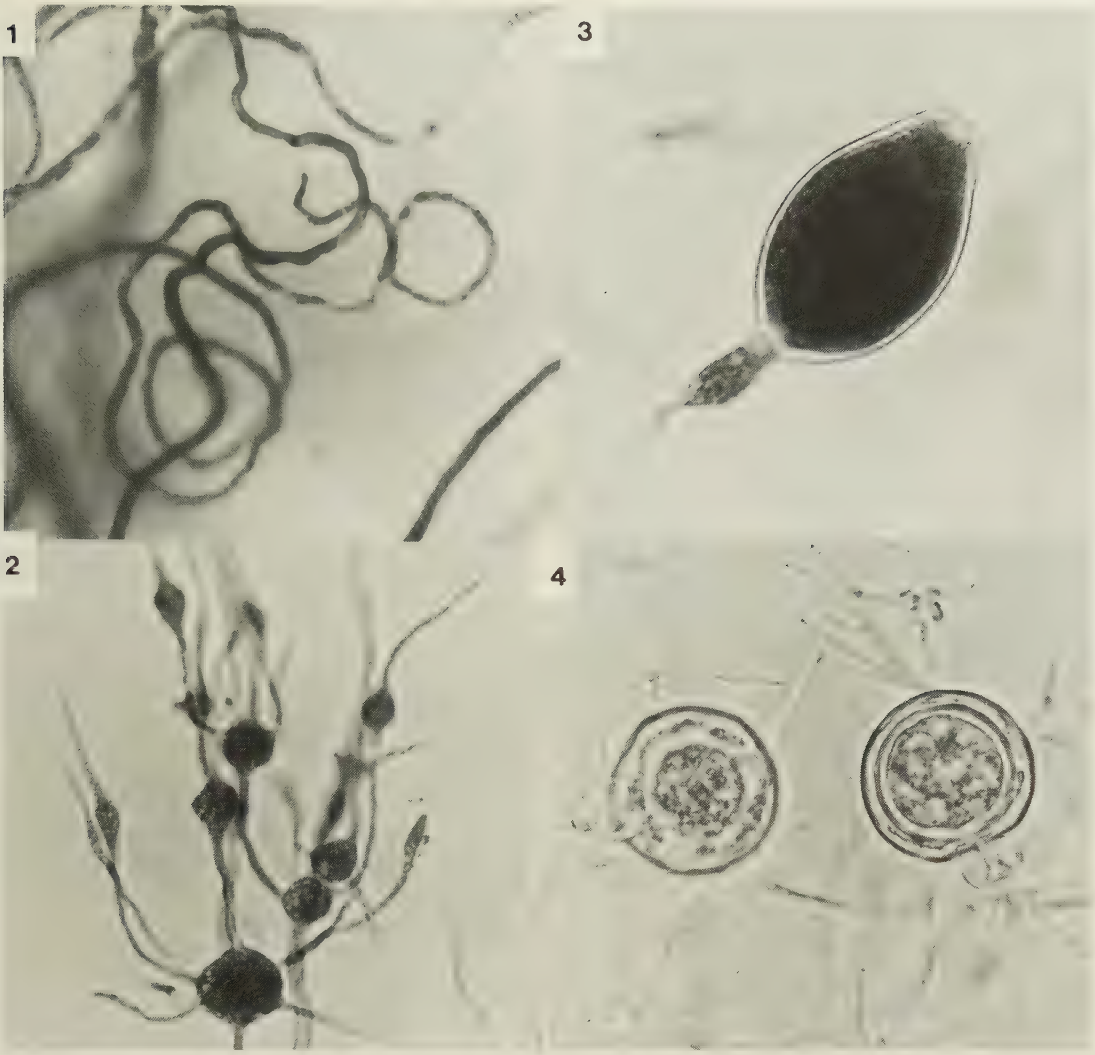
290 Lateral lesion on leek

OCCURRENCE White tip disease has been reported in the UK on spring onions (3) and leeks (2), as well as on onions and shallots. In the Netherlands it occurs on leeks (1,4) and on onions (8), and in Japan on leeks, scallions, onions and garlic (5). In South Africa it was recorded on onion seedlings under sprinkler irrigation (7).

SYMPTOMS The tips of the leaves die back for several cm and turn white; leaves are deformed, and water-soaked areas develop towards the base of the leaves (3,4). A serious rot may develop on onion bulbs in store (10).

BIOLOGY The fungus persists in the soil, producing both the asexual stage (sporangia which may give rise to zoospores) and the sexual stage (thick-walled oospores capable of surviving unfavourable conditions) (2,3,9). Resting spores (chlamydospores) may also be produced (6). Infection probably occurs from wind-borne sporangia (2) or water-splashed zoospores.

CONTROL Crop rotation is advisable (10). Studies of temperature and rainfall permit disease forecasting (10), so that fungicide sprays may be appropriately timed (1,4,8).



291 *Phytophthora porri* (from CMI Descr. No. 595, Stamps 1978) 1 hyphal coils on agar 2 hyphal swellings in water 3 sporangium 4 oogonia with paragynous and amphigynous antheridia × 500

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1 BAKEL J.M.M. van (1964) Papiervlekkenziekte in prei (*Phytophthora porri* F.). *Mededelingen van de Directeur Tuinbergen* **27,** 198–200 illus.

2 FOISTER C.E. (1931) The white tip disease of leeks and its causal fungus, *Phytophthora porri* n.sp. *Transactions of the Proceedings of the Botanical Society of Edinburgh* **30,** 257–281.

3 GRIFFIN M.J. & JONES O.W. (1977) *Phytophthora porri* on autumn-sown salad onions. *Plant Pathology* **26,** 149–150 illus.

4 HOOF H.A. van (1959) Oorzaak en bestrijding van de papiervlekkenziekte bij prei. *Tijdschrift over Plantenziekten* **65,** 37–43.

5 KATSURA K., ISAKA M. & MIYAGOSHI M. (1969) [*Phytophthora porri* Foister, the causal fungus of the leaf blight and bulb rot of scallion, *Allium bakeri* Regel.] *Annals of the Phytopathological Society of Japan* **35,** 55–61.

6 KATSURA K. & MAEDA S. (1974) [Chlamydospore formation of *Phytophthora porri* Foister.] *Scientific Reports of Kyoto Prefectural University, Agriculture* **23,** 34–38.

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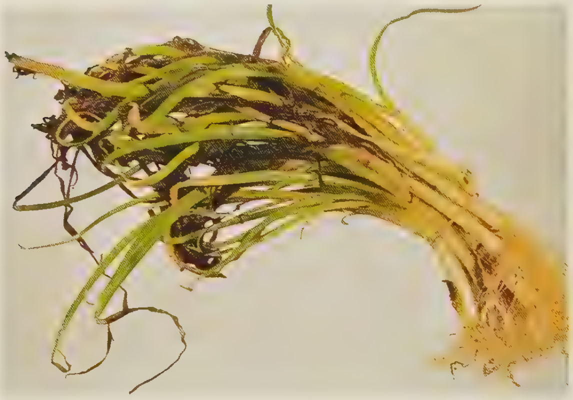
10 YOKOYAMA S. (1976) [Studies on the leaf blight of onion (Tamanegi Shiroiro Ekibyo) caused by *Phytophthora porri* Foister.] *Bulletin of Fukuoka Agricultural Experiment Station* No. 22, 55 pp.

OTHER DISEASES of spring onions, leeks and chives

BACTERIAL BLIGHT of leeks is reported from France (8) and New Zealand, the causal organism being *Pseudomonas syringae* (page 112). Oval watersoaked lesions are produced on the leaves.

BACTERIAL ROTS may be caused by *Erwinia carotovora* ssp. *carotovora* (page 271) and *Pseudomonas fluorescens* (3).

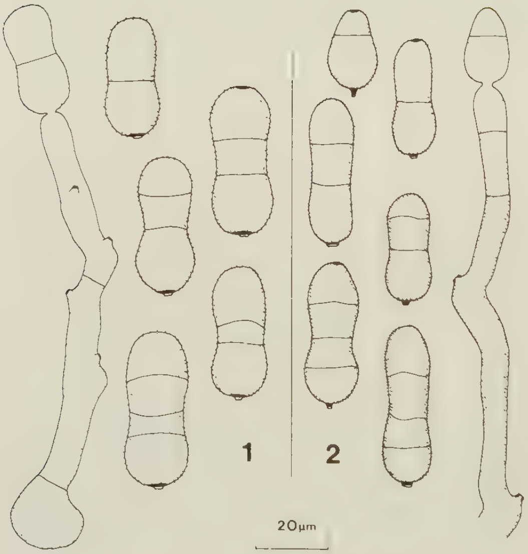


292 Grey mould rot of spring onions

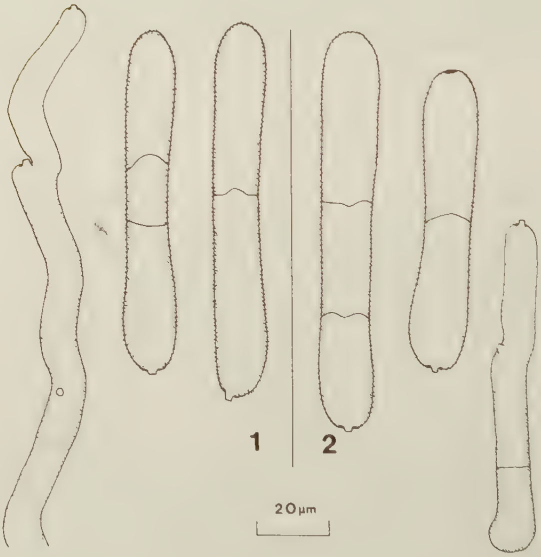
BLACK LEAF STRIPE of leeks, caused by *Leptotrochila porri* v. Arx & Boerema, was recorded first in the Netherlands and subsequently in New Zealand, probably having been transmitted on Dutch seed (9). Black streaks also form on the outer scales of the stem base, and market value is reduced.

DOWNY MILDEW, caused by *Peronospora destructor*, can occur on spring onions and leeks; leaf symptoms are described under Diseases of Onions, Shallots and Garlic (pages 246, 247).

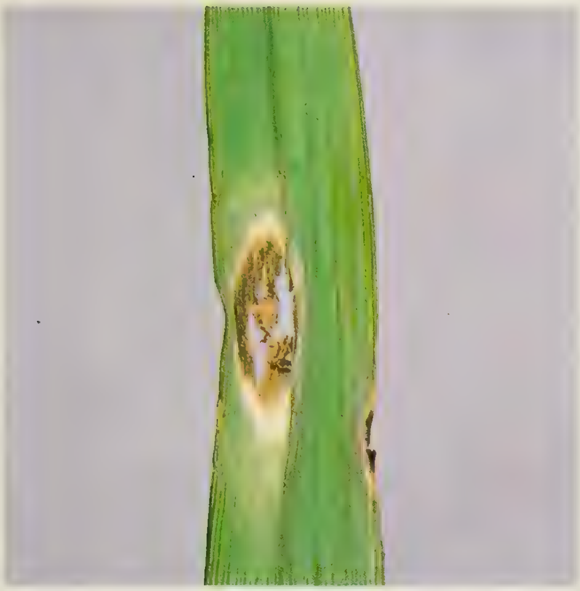
GREY MOULD ROT and **LEAF BLAST** can be caused by any of several species of *Botrytis*, but mainly *B. allii* (page 251). A rot of the fleshy leaf bases can occur (2), or there may be leaf-spotting and tip dieback followed by complete blighting of the foliage. The fungus is considered in more detail under Diseases of Onions, Shallots and Garlic (page 250). *Botrytis porri* Buchw. causes an important storage disease of leeks in Scandinavia and has also been recorded in the UK. The symptoms are pale brown watersoaked lesions on the inside of the leaves and leaf bases, and the fungus is capable of slow growth even at 0°C (10). Control is achieved by means of appropriate fungicide sprays.



293 *Cladosporium allii* (from CMI Descr. No. 841, 1986) Conidiophore and conidia 1 from the host 2 from culture



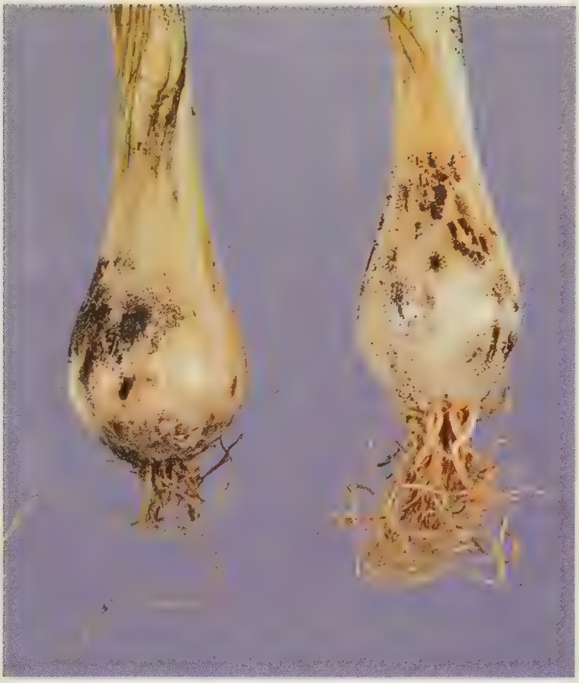
294 *Cladosporium allii-cepae* (from CMI Descr. No. 842, 1986) Conidiophore and conidia 1 from the host 2 from culture



295 Cladosporium leaf blotch of leek

LEAF BLOTCH of leeks is caused by *Cladosporium allii,* while the corresponding species on onions is *Cladosporium allii-cepae* which overwinters in the sexual state, *Mycosphaerella allii-cepae* (5,6).

PLEOSPORA ROT of leeks, caused by *Pleospora herbarum* (page 211) has been reported from the Netherlands, following wet weather (1). Oval lesions develop on the leaves; affected tissue is initially soft but later dry and brownish white. Under humid conditions the sexual state (pseudothecia) develops after about 2 months (1). The disease can be controlled with fungicide sprays.



296 Smudge of spring onions

PURPLE BLOTCH, caused by *Alternaria porri* (Ell.) Cif., can cause onion crop losses, and the disease has also been recorded on leeks in the UK (4) and the USSR. It is considered in detail under Diseases of Onions, Shallots and Garlic (page 252).

SMUDGE of leeks, described as a leaf blight, has been reported from India (7). The causal fungus is *Colletotrichum circinans,* considered in detail under Diseases of Onions, Shallots and Garlic (page 256).

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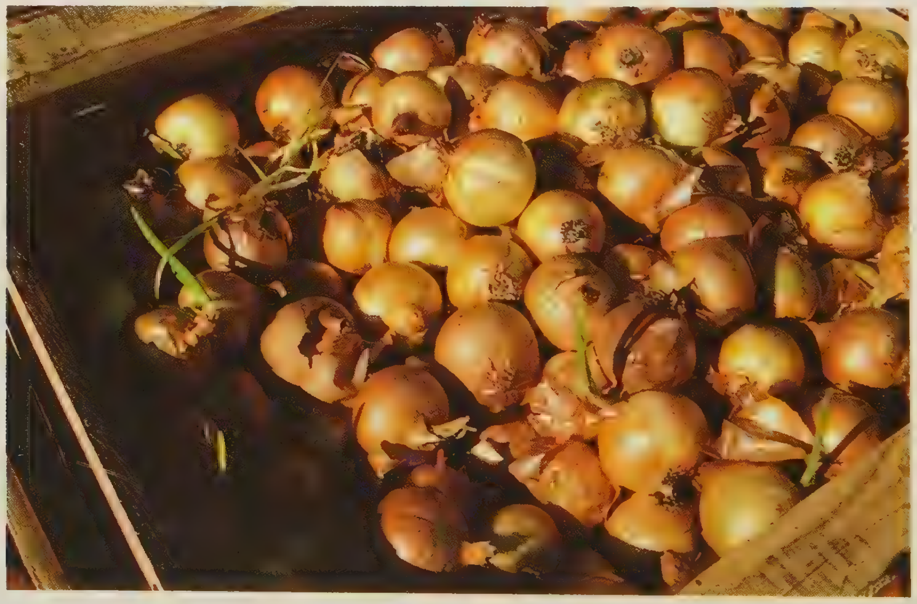
CHAPTER 7
BULBS

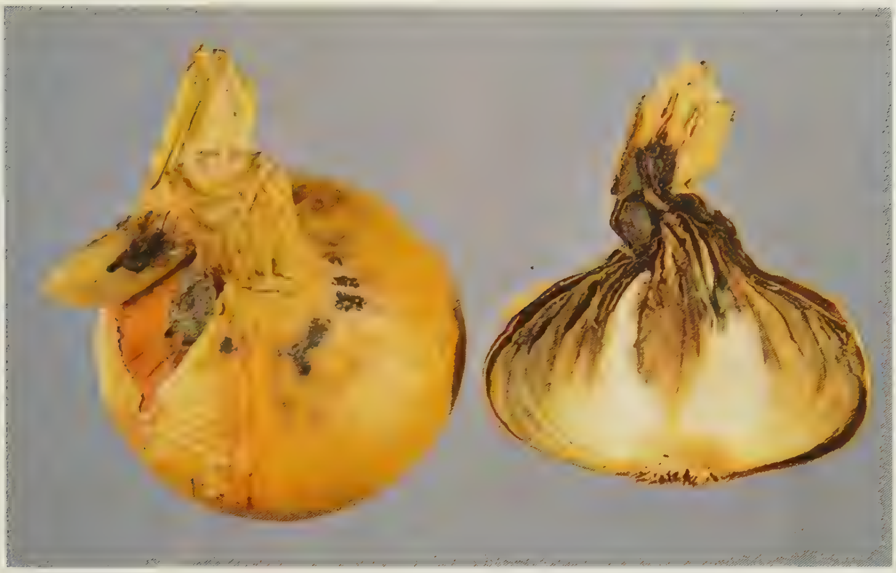
Both **onion**, *Allium cepa* L., and **garlic**, *Allium sativum* L., are thought to have originated in central Asia. They have been cultivated in India and China since very early times, and were highly esteemed in ancient Egypt, Greece and Rome (3). Onions are now grown in most countries of the world, and total production exceeds 25 million tonnes. The leading producers are China, India, the USSR, the USA, Turkey, Japan, Spain, Brazil, Iran and Poland. Substantial quantities are exported from the Netherlands, Spain, India, Turkey and Poland, the greatest demand being in West Germany and the UK. World production of garlic is about 2½ million tonnes, grown chiefly in China, Korea, Spain, India, Egypt, Thailand, Turkey, Brazil, the USA and Indonesia.

Allium species are monocotyledonous plants possessing long green leaves. Some, for example spring onions, leeks and chives, are eaten young and fresh; they are considered in Chapter 6. The present chapter is concerned with cultivars grown for their mature bulbs, which are storage organs consisting of swollen leaf bases covered by thin scales (1). Colours vary from white through brown to reddish purple (3). **Shallots**, characterised by clusters of small bulbs joined at the base, are no longer considered a distinct species but simply a variety of onion, *Allium cepa* var. *aggregatum*. A head of garlic is composed of several bulblets or 'cloves' which together are enclosed in an outer papery scale. Garlic and shallots are propagated vegetatively, while many onion cultivars can be grown from seed as well as from 'sets' (small bulbs grown specially for the purpose). The onion is a biennial plant, producing a storage bulb in the first year and, if permitted, a flower head in the second (1). Bulbing is controlled by day-length and temperature, and different cultivars are appropriate for temperate and tropical latitudes (3).

Harvested bulbs (unless intended for immediate consumption) are subjected to a 'curing' process in which the most important feature is removal of moisture from the outermost layers (7,10). The purpose of curing is to reduce the likelihood of fungal or bacterial infection, and a well-cured onion has a tight neck and crisp, rustling outer scales. In countries with a hot dry season the crop may be cured in the field, the pulled bulbs being covered by green tops of the adjacent row, as protection against direct sunlight (3). Field curing is completed in a matter of days, the length of time depending on the maturity of the onions when pulled (8) and the atmospheric temperature, humidity and wind speed. In countries with an unreliable climate it is advisable to have an artificial means of drying. The onions are topped and gathered into store on the same day, and then the base of the stack is subjected to a blast of hot air (30°C), the aim being rapid removal of surface



297 Sprouting of onions



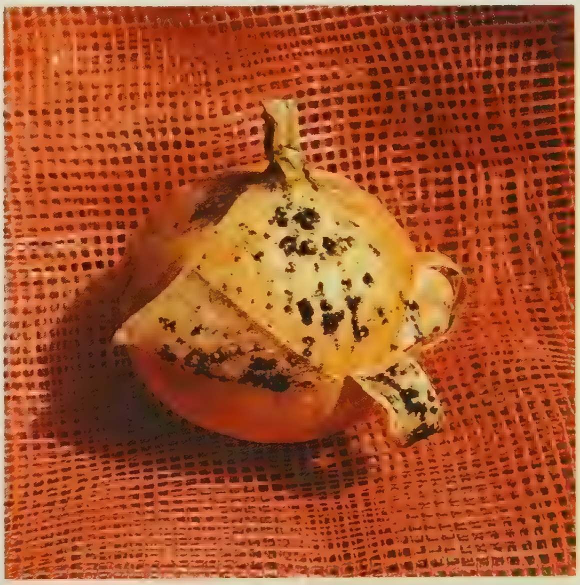
298 Grey mould and neck rot of onions

moisture from the bulbs (7). When the onions at the top of the stack are surface-dry the rate of airflow is reduced, but the drying process is continued for about 4 weeks until the cell sap moisture in the onion necks has evaporated, leaving them tight and dry (7,10). Brown-skinned cultivars take on an attractive golden colour if cured at high temperature, but at 30°C and above there is a risk of proliferation of black mould (q.v.). Consequently it is important to lower the temperature to about 27°C after the first 2 or 3 days (7).

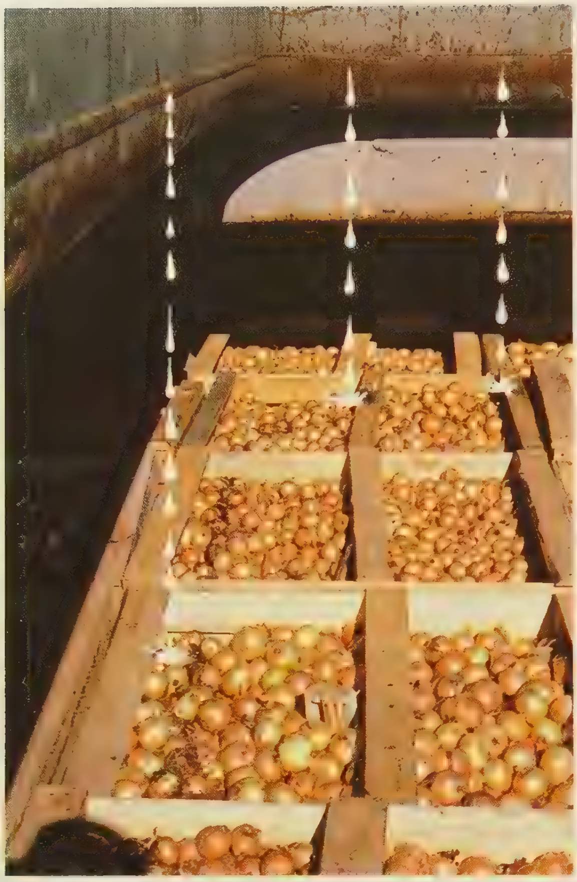
After the curing period is completed, the onions should be kept as cool as possible. Indeed it is sometimes advantageous to store them slightly below their freezing point (supercooled but not frozen), although care must then be taken to avoid any vibration (which would result in instant freezing) and the onions must be allowed to warm up before being moved.

Even more important than temperature is the relative humidity of the storage atmosphere (7). Onions differ from most other horticultural commodities in requiring a relative humidity of only 70 to 75%, and this presents a problem which is not easily overcome. Since one of the products of respiration is water vapour, the atmosphere within a mass of any fresh produce tends to be very moist, and the relative humidity is commonly above 90%. In a modern refrigerated onion store, therefore, the air is first of all cooled to well below the required storage temperature, as a means of inducing excess water to condense out. This partially dried air is then re-heated to the required temperature and blown through the onions. The desired relative humidity can be maintained by means of a humidistat, and a compromise of 75 to 85% is usually considered satisfactory – a lower value is not worthwhile for economic and practical reasons. Under such conditions onions may be stored for up to 10 months. A few days before removal from refrigerated store, the onions should be allowed to warm up gradually to a temperature above the dew-point of the outside air, otherwise atmospheric moisture would condense on the cold bulbs. Similar remarks apply to garlic.

Onions and garlic are frequently stored at ambient temperature. In temperate climes they must be protected from winter frosts, while in the tropics both temperature and humidity are often unavoidably high (5,10). Temperatures above 25°C inhibit sprouting (q.v.) but encourage decay.



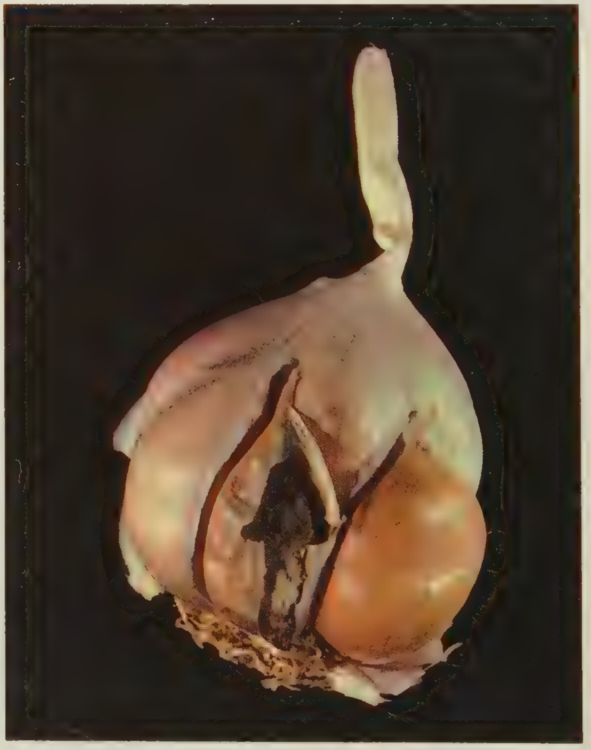
299 Black mould rot of onion



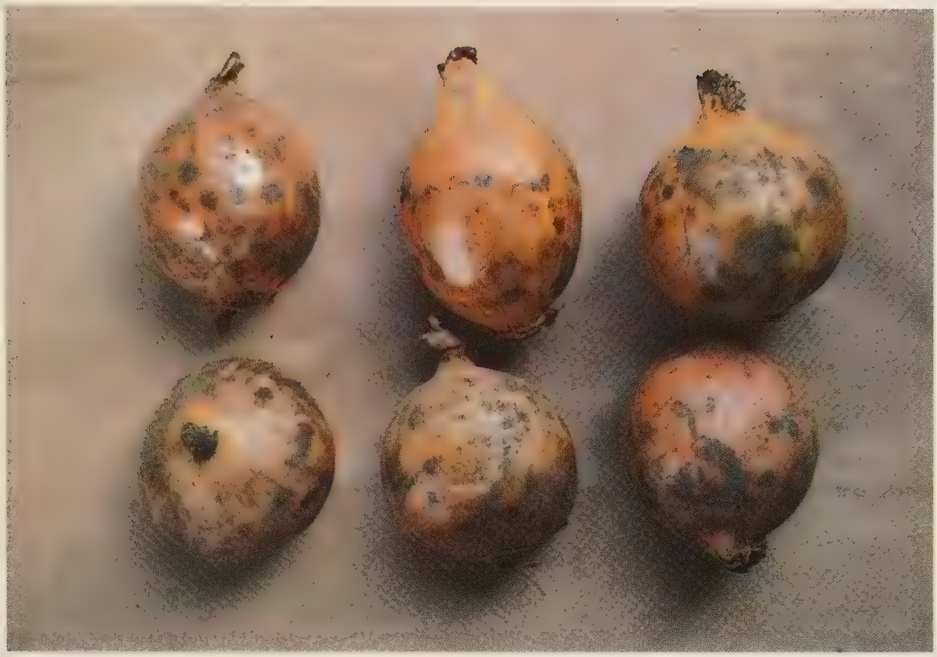
300 Water-staining on board ship (condensation from metal hatch-coaming)

The transport of onions and garlic presents even greater problems than storage on land. In a refrigerated ship the hatch temperature can be finely adjusted, but it is impossible to achieve a low enough relative humidity. The best that can be done is to reduce the functional area of the cooling coils so that they need to be run at a lower temperature to achieve the given air delivery temperature. A sufficiently low coil temperature results in copious amounts of water vapour condensing out of the air and freezing on the coils; despite the necessity for defrosting, this procedure is desirable as a means of reducing the amount of moisture in the circulating air. An important factor influencing moisture content is the quantity of fresh air introduced into the hold. Refrigerated onions respire slowly and do not require large quantities of fresh air, so if the vents are opened for about one hour nightly this is probably sufficient. To prevent atmospheric moisture condensing on the bulbs during unloading, the cargo should be allowed to warm up during the last few days of the voyage, until its temperature is above the dew-point.

It is more usual for onions and garlic to be carried in ventilated vessels, and it is generally recommended that the fans be capable of 15 to 20 volumes per hour. Shallow decks are preferable, so that the height of the stow is not excessive; this is especially important if the onions are packed in nets, as these are difficult to ventilate, tending to bed down into a closely packed mass. Air channels should be constructed fore-and-aft and athwartships, maintained by wooden planks (dunnage), and the stow should be raised off the deck. If the onions are packed in wooden cases, air circulation is facilitated. If they are packed in wooden bins there may be a danger of self-heating in the centre, and for this reason some packers construct their bins with a vertical chimney framework in the middle to permit ventilation of the interior. The fact remains that at sea the available air is usually moisture-laden, and consequently there is much debate about the correct procedure for ventilating an onion stow during a voyage through several climatic zones. The main concern is the danger of atmospheric moisture condensing on the metal deckhead and dripping on to the onion stow. Some shippers therefore give instructions that the dew-point be ascertained at regular intervals, and that the fans be switched off whenever the hatch temperature is lower than or equal to the dew-point. However, it is considered that far more risk attaches to interruption of ventilation than to condensation – the onions themselves continue to produce moisture, and if ventilation is stopped it will not be long before saturation point is reached within the stow. It is



301 Blue mould rot of garlic



302 Close-up of water-stained bulbs

therefore recommended that the fans be run throughout the voyage, except perhaps during heavy weather if there is risk of sea-spray entering the vents.

A considerable amount of work has been done on the modification of 20-foot containers to make them suitable for the ventilated carriage of onions (6). 'Ventainers' rely on natural ventilation. 'Fantainers' are supplied with a fan which may be mounted in an exhaust vent near the top of the forward end, with small inlet vents at the bottom of each long side; alternatively, a fan is mounted on the floor at the rear, and one of the container doors is removed. Whatever the system, it is important to ensure that the onions are properly stowed, with plenty of dunnage to facilitate air circulation between and beneath the bags (6).

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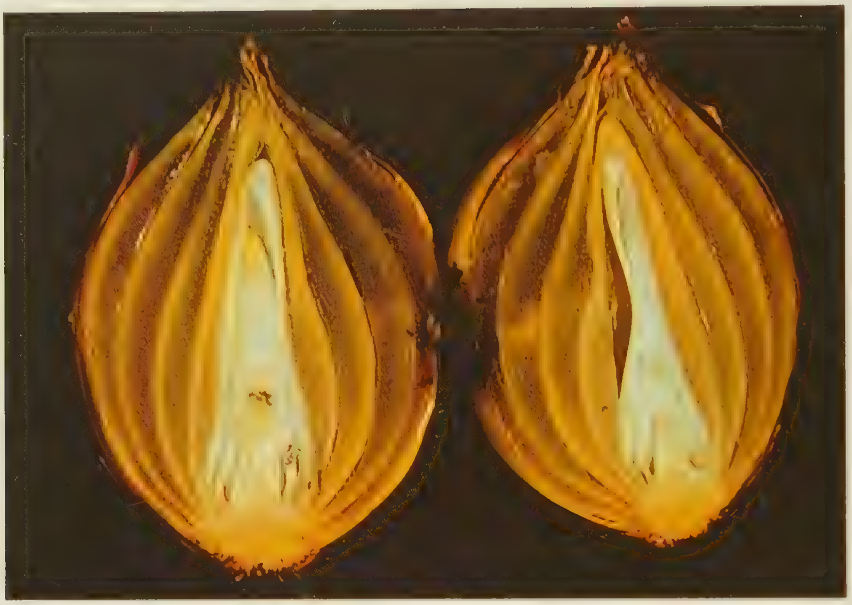
BACTERIAL ROTS of onions, shallots and garlic caused by

Erwinia **spp.**
Lactobacillus **spp.**
Pseudomonas **spp.**



303 Rotting caused by *Lactobacillus* sp.

Symptoms vary but, in contrast to the fungal rots, bacterial rots often tend to progress within the affected fleshy scale rather than moving directly into adjacent fleshy scales. Decay may be caused by a complex of several species, making it difficult to identify the primary organism (3,10).

Erwinia carotovora **ssp.** ***carotovora*** (page 271) has caused serious losses of onions in the USSR and of garlic in Egypt (1). Infected bulbs may appear sound externally, the only sign of disease being an unpleasant odour; pressure on the bulb may cause exudation of foul-smelling juice from the neck. Diseased tissues are soft and slimy.

Erwinia chrysanthemi **Burkholder** is reported on onions in Australia (3), inciting a cream-coloured soft rot which develops rapidly under warm conditions.

Erwinia herbicola **(Löhnis) Dye** has been recorded on onions in the UK (4), causing a firm watersoaked rot with no distinctive odour.

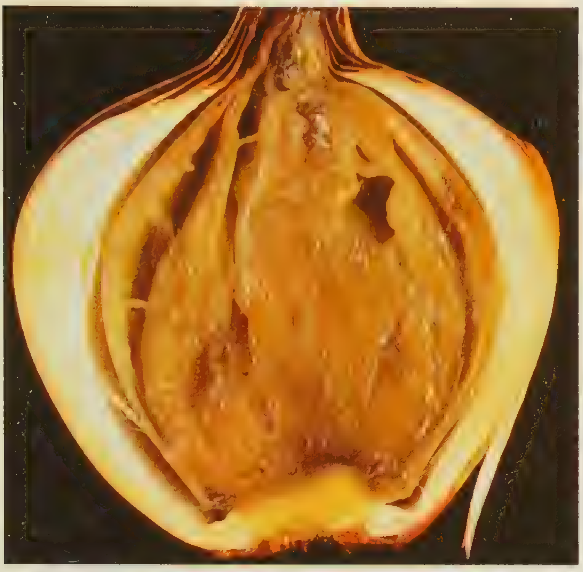
Erwinia rhapontici (page 220) causes a soft rot of onions during cool seasons in Japan (5).

Lactobacillus **spp.** are ubiquitous, and can cause serious rotting at temperatures above 30°C (4). The inner fleshy scales are attacked; infected tissue appears pickled, and emits an odour of vinegar.

Pseudomonas aeruginosa **(Shroeter) Migula** has caused serious storage losses in Australian onions, following a wet growing season. Initially the decay is confined to one or two of the inner fleshy scales, which turn brown but remain firm (3,7). Softening of the neck area may be caused by the entry of secondary bacteria. Eventually the bulb undergoes collapse and the exuded juices cause infection of adjacent bulbs.

Pseudomonas cepacia **Palleroni & Holmes** has been recorded on onions in Italy (2), the USA (8) and Papua New Guinea (9). This bacterium characteristically attacks certain of the outer fleshy scales (never the innermost ones) (2) and induces the formation of a yellow slimy substance. The disease was given the name of 'sour skin' because of the characteristic odour and because of the location of the rot, or alternatively 'slippery skin' because slight pressure causes the healthy inner part of the bulb to slip out. Subsequently, however, the name 'slippery skin' has been used in reference to the symptoms caused by *P. gladioli* pv. *alliicola* (q.v.).

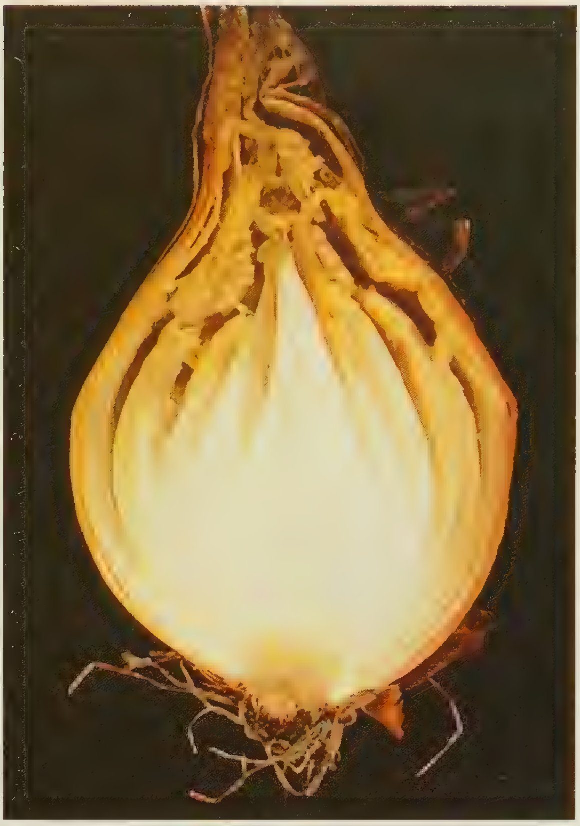
Pseudomonas gladioli **pv.** ***alliicola*** **(Burkholder) Young** ***et al.*** has been recorded on onions in the USA, Australia (3,7), the UK (4), and on Spanish onions imported into the UK (6). It typically causes a watersoaking of the inner fleshy scales, although the entire bulb may rot eventually (6). Infected tissue is mushy and may have a greenish tinge.



304 *Pseudomonas gladioli* pv. *alliicola*

Healthy bulbs may contain low levels of bacteria which normally cause no damage but which can attack tissue weakened by environmental stress such as excessive heat (3,4). Bacteria are transmitted by water, and disease incidence has been related to rainstorms, flooding and irrigation (8). Wounding is a common predisposing factor, and infection can occur before or during harvest (3,8).

An important control strategy is to avoid cutting too closely during 'topping' of the bulbs; it is preferable to make the cut several cm above the neck (4). Drying should be carried out promptly, at temperatures not exceeding 30°C, and the store temperature should be reduced as soon as curing is completed (4).



305 *Pseudomonas gladioli* pv. *alliicola*

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BLACK MOULD ROT of onions, shallots and garlic caused by
Aspergillus niger v. Tieghem

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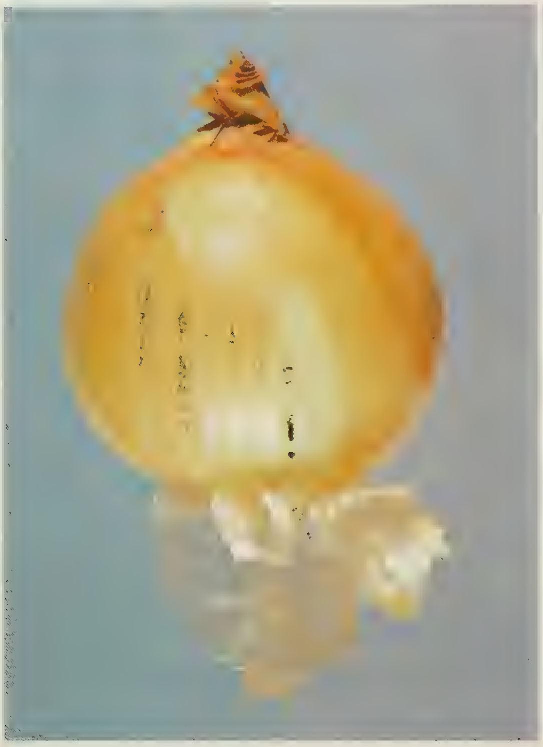

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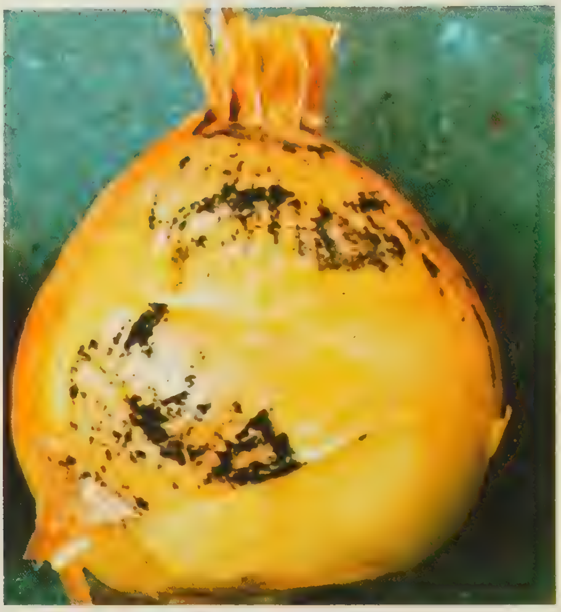

OCCURRENCE This fungus is widely distributed but thrives only at high temperatures. It is therefore of importance in tropical regions such as the Sudan (1) and India (3,8), and in countries with a hot growing season such as Australia (6), Japan (10), Spain (4), Chile (2) and the southern states of the USA (5). As a result of the introduction of high temperature drying techniques it has appeared in onion stores in the UK (9).

SYMPTOMS Clusters of black spores form on and between the outer papery scales, generally aligned along the veins. Black mould causes a shrivelling of the affected papery scales, and may advance into the fleshy scales accompanied by soft rot bacteria. At high temperatures and in humid storage conditions spore production on the exterior of the bulb may be so profuse as to resemble soot.

BIOLOGY The fungus can persist in the soil and on crop debris, and its spores are blown about in the atmosphere. Infection of bulbs is thought to take place usually via the neck tissues when the foliage dies down at maturity (4). Rain around harvest-time has been associated with high levels of infection (4), but this fungus can also cause infection under dry conditions (7).

CONTROL Black mould can be transmitted in seed (3) or on sets, and it may therefore be beneficial to carry out a pre-planting fungicide treatment (3). In irrigated crops it can be advantageous to stop the water supply approximately 3 weeks before harvest. An application of lime to the topped bulbs has been found to reduce losses in store (10); alternatively a fungicide dip (6) or a fumigant (8) can be used. The main post-harvest measure, however, is prompt and appropriate drying after harvest; it is important that the curing temperature does not exceed 30°C and that the relative humidity does not exceed 80%. The temperature should be reduced to below 25°C within 2 or 3 days. Refrigerated storage arrests the development of black mould completely, although fungal growth is resumed on return to ordinary temperatures.

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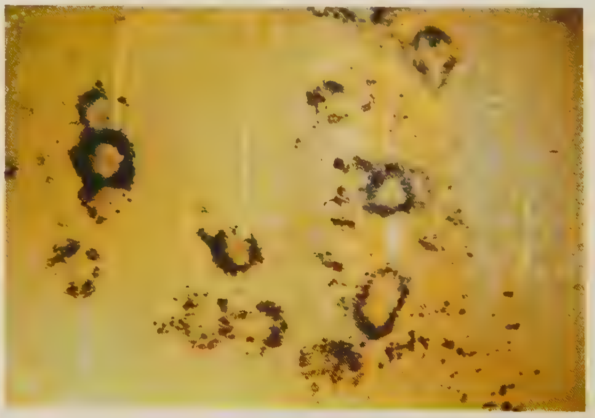
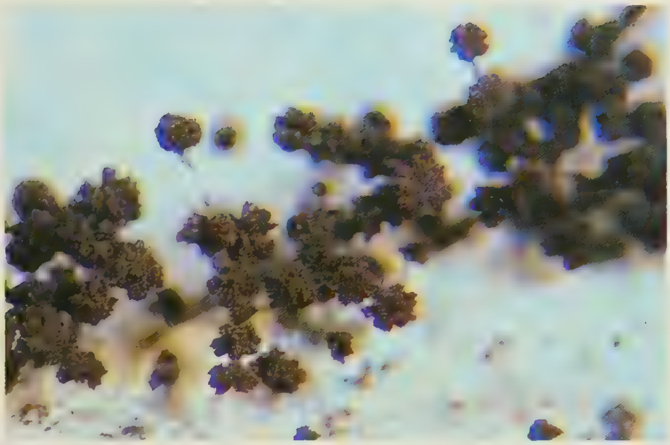
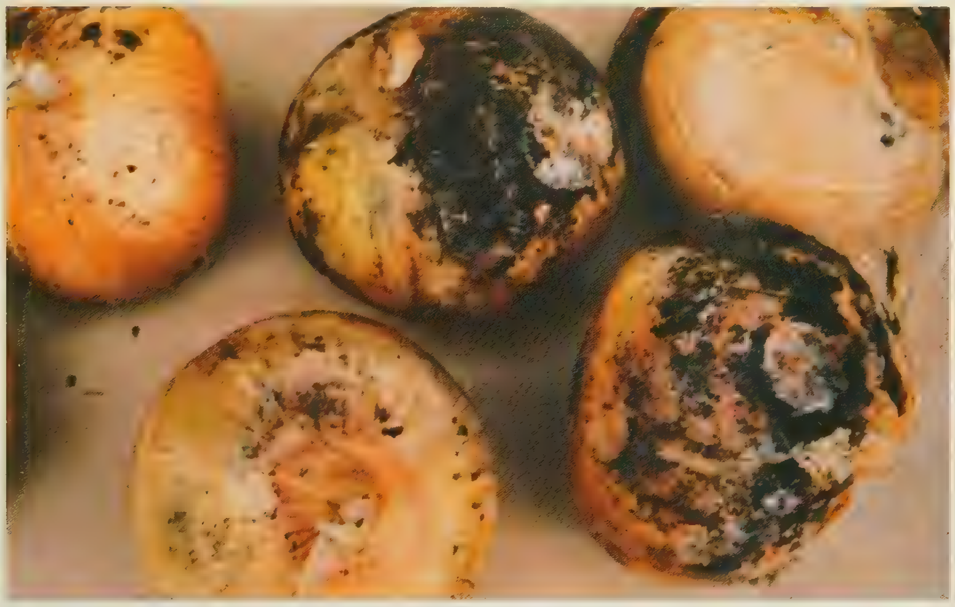




309 Conidial heads of *Aspergillus niger*



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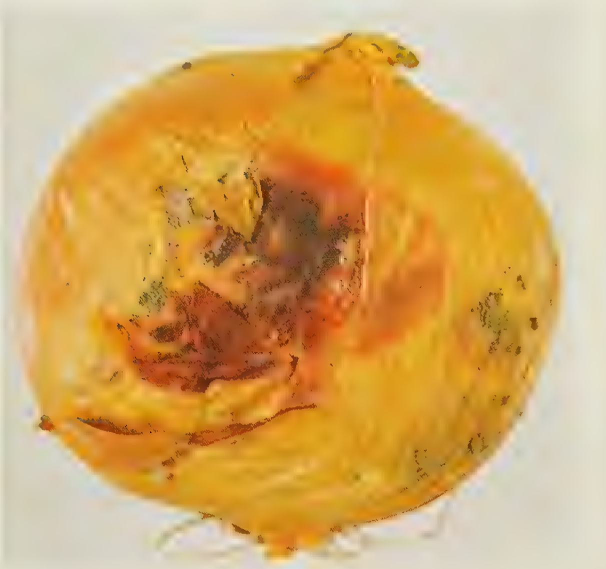
BLUE MOULD ROT of onions, shallots and garlic caused by *Penicillium* spp.

311



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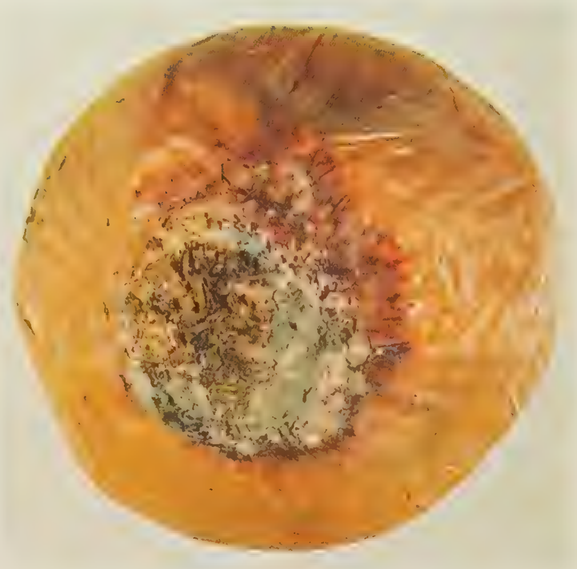

OCCURRENCE Blue mould rot, caused by various species of *Penicillium*, has been recorded on stored onions in New Zealand (2), the UK, Finland and Germany (7), in Australian, Spanish and Dutch onions imported into the UK (10) and in stored shallots in Denmark, generally as a minor cause of loss. In garlic blue mould rot, usually caused by *P. corymbiferum* Westling, has led to serious losses, for example in the US (1,9), Chile (3), Colombia (8) and Bulgaria (6).

SYMPTOMS The earliest symptoms are pale yellowish lesions, but these are soon covered with characteristic blue-green spores (3). In onions and shallots the disease may exist only as a blemish although some species, for example *P. cyclopium* Westling, can cause a soft watery rot (10). *P. purpurescens* (Sopp) Raper & Thom (which is frequently associated with Tasmanian onions) produces a striking purplish red pigment which stains the scales. In blue mould rot of garlic (sometimes called green rot) (5), the contents of entire cloves may be converted to a lightweight mass of spores (3).

BIOLOGY In a detailed study of *P. corymbiferum* on Californian garlic (9) it was established that the fungus could not survive long in soil, and that it was therefore probably transmitted on planting material. Infection occurred via wounds sustained when the cloves were separated by breaking apart the parent bulb in preparation for planting, and rotting was initiated in the field.

CONTROL Control measures include prompt drying (curing) of harvested bulbs, followed by fungicide treatment (9), and chemical treatment of the soil before planting (6). Some cultivars show resistance (4).

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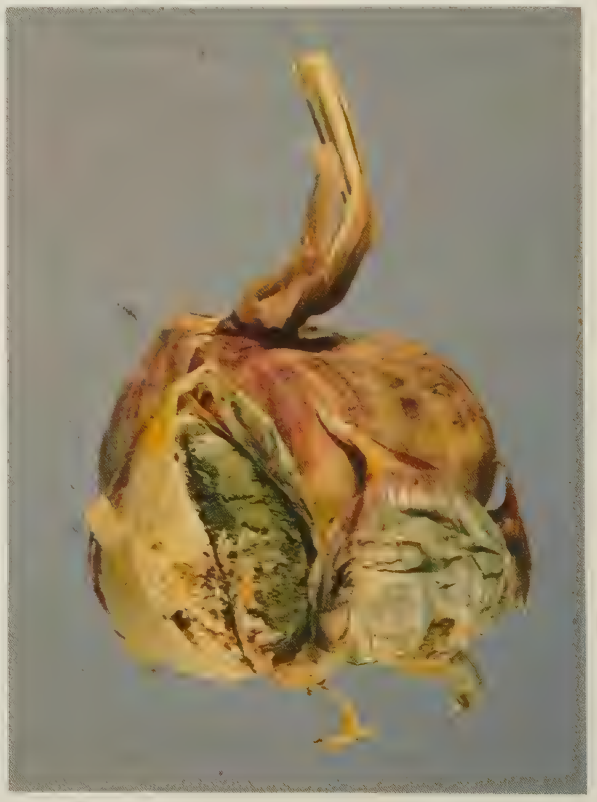
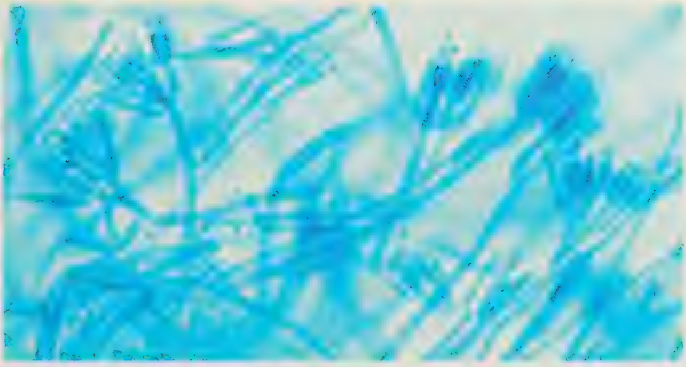
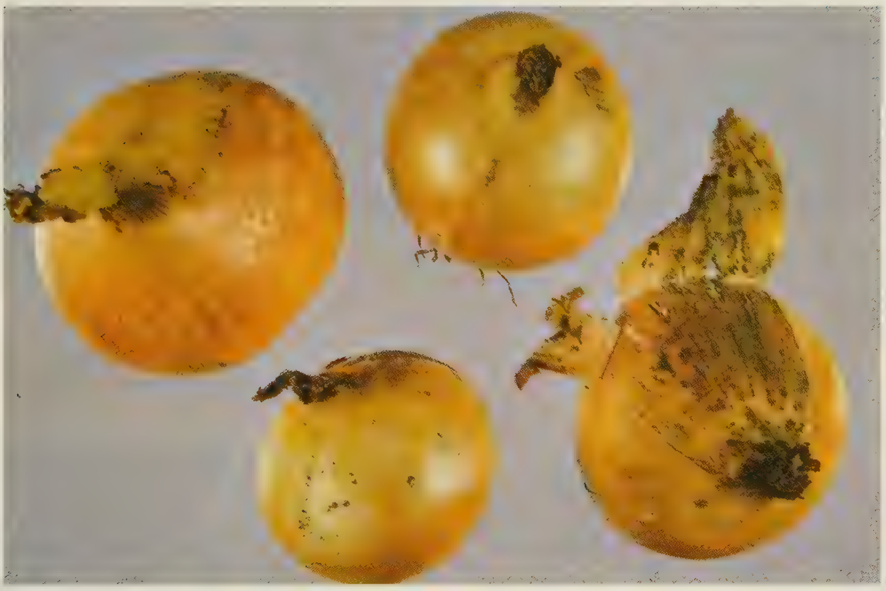




314 Conidial heads of *Penicillium italicum* (stained with cotton blue in lactophenol) × 400



315

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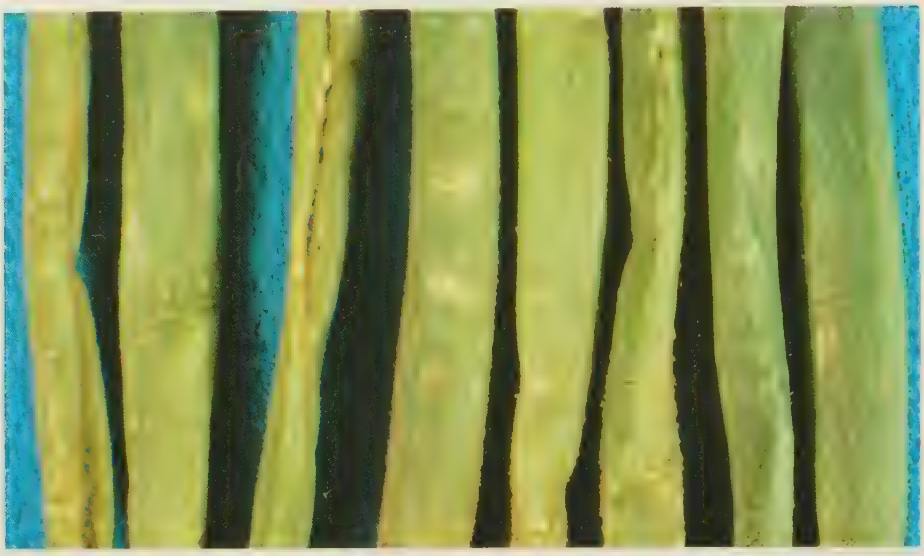
DOWNY MILDEW of onions, shallots and garlic caused by ***Peronospora destructor*** **(Berk.) Casp. ex Berk.**

316



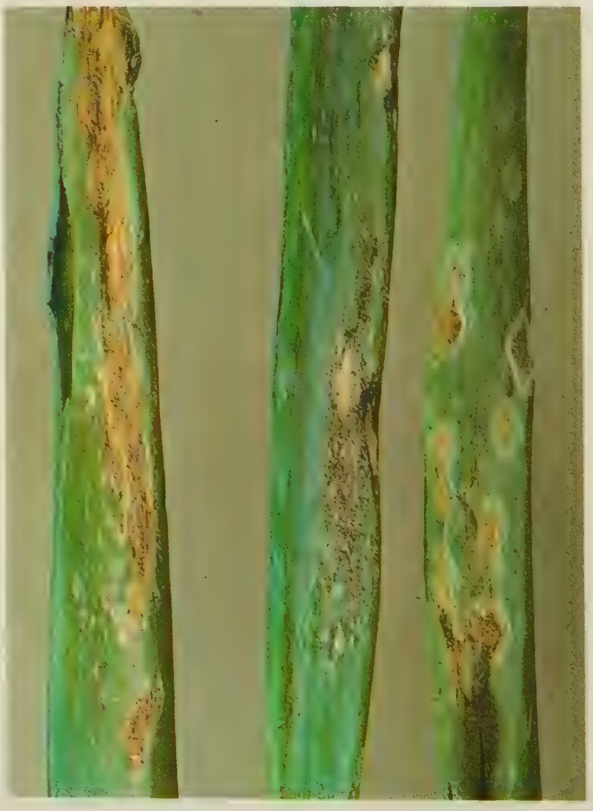
OCCURRENCE Downy mildew is mainly important in regions having a cool moist climate, for example the UK, New Zealand (9), Japan and the USSR (7), but the disease can also be of significance during the cool season in subtropical countries, for example northern India (5), Israel (6), Brazil (2) and the southern states of the USA (10).

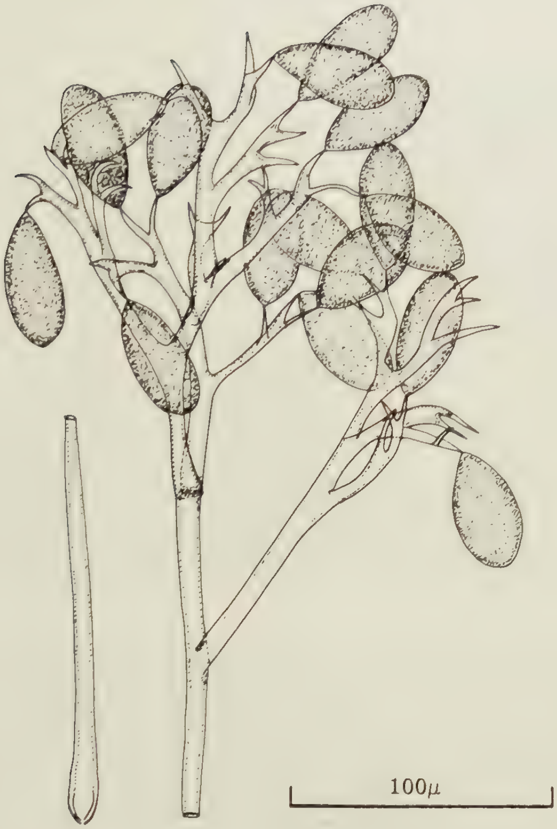
SYMPTOMS An infected bulb becomes soft and shrivelled in store, and the outermost fleshy scale is discoloured, wrinkled and watery. The basal plate is permeated with fungal strands, and the bulb may be induced to sprout prematurely.

BIOLOGY The fungus produces both the asexual stage (lemon-shaped sporangia) (4) and the sexual stage (thick-walled oospores) (7) and either type of spore can cause infection of growing plants by being blown or splashed up from the soil (1). White spots are produced on the leaves, and if humid conditions persist there is copious production of purplish-grey spores on the lesions (10). The optimal temperature for disease development is approximately 12°C (1, 10) and outbreaks have been correlated with rainfall and dew formation. Disease development may also be dependent on cultural as well as climatic factors; plant density, row orientation and method of irrigation determine whether or not the foliage remains wet long enough for infection to occur (6).

CONTROL The fungus is transmitted in seed and in sets (1,7). There may be no obvious symptoms in diseased planting material (4), but it would be advisable to discard bulbs which have begun to sprout prematurely. Fields should be well-drained and free of weeds; volunteer plants should be destroyed (3). Forecasting systems (3,6) permit timely spraying of the growing crop with an appropriate fungicide (2,5,8,9). Trickle irrigation is preferable to sprinkle irrigation (6).

317





318 *Peronospora destructor* (from CMI Descr. No. 456, Palti 1975) Sporangiophore and sporangia (conidia)

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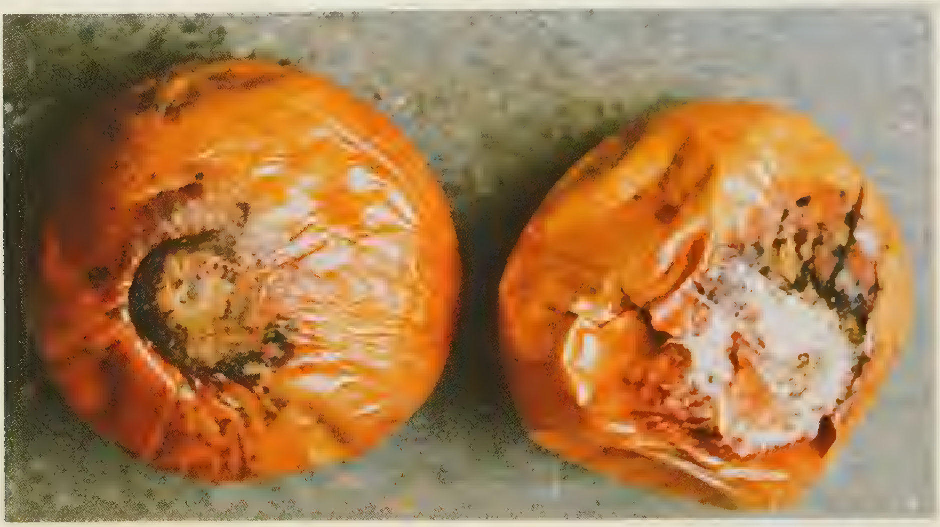
FUSARIUM BASAL ROT of onions, shallots and garlic caused by ***Fusarium oxysporum*** **f.sp. *cepae*** **(Hanz.) Snyder & Hansen**

319



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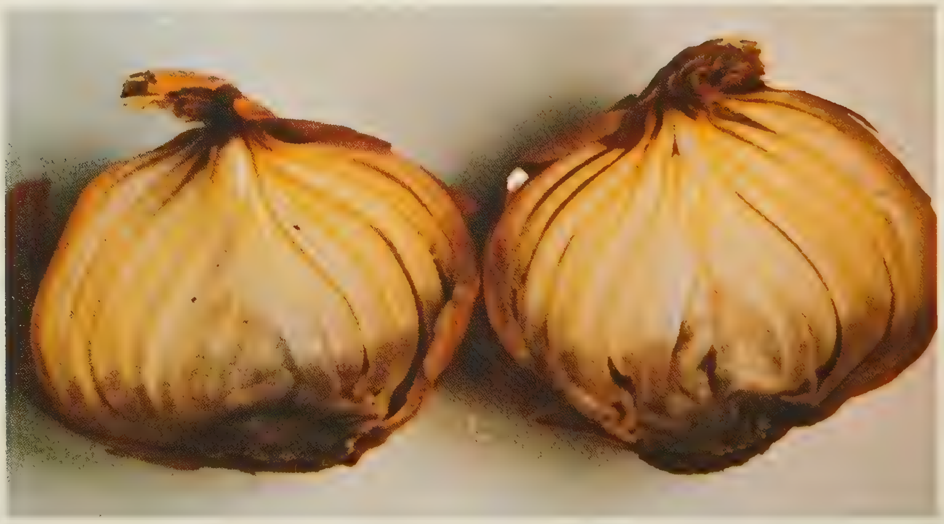
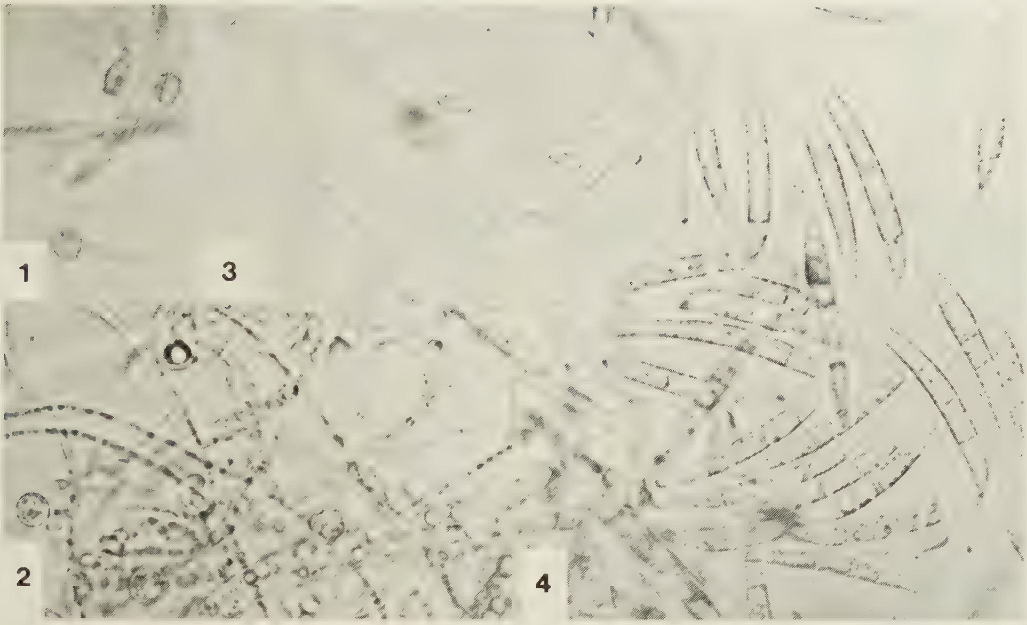


OCCURRENCE Basal rot occurs in most countries but tends to be associated with hot growing conditions (3). *F. oxysporum* f.sp. *cepae* has been recorded on onions in, for example, South Africa (5), Italy (4), Japan (6), and many parts of the USA (2,3, 7,10). Garlic may be attacked by the same fungus (9), though in the western USA it is *F. culmorum* (W.G. Smith) Sacc. which has been identified as the cause of major losses in the California and Oregon garlic industry (1).

SYMPTOMS Symptoms in stored bulbs include a mass of white mould, sometimes accompanied by a pinkish tinge at the base of the bulb; the roots may have already have rotted away. A semi-watery decay progresses in a broad front up the bulb (10); during prolonged storage the bulb shrivels and becomes a lightweight 'mummy'. In garlic the cloves tend to 'shatter' from bulbs with damaged basal plates (1).

BIOLOGY The fungus is present in the soil, surviving unfavourable periods in the form of thick-walled chlamydospores (3). It attacks the growing crop, resulting in reduced yields (1). Infection occurs via the roots and stem-base (10), and penetration may be direct, it may (7) or may not (3) occur via injuries caused by maggots, or it may follow in the wake of the pink root disease organism (2,9) (page 258). Disease severity has been linked with high temperatures following heavy rains, flood irrigation or, conversely, low soil moisture (3,6).



321 *Fusarium oxysporum* (from CMI Descr. No. 211, Booth 1970) 1 chlamydospores formed in cells of macroconidia 2 hyphal chlamydospores 3 microconidia 4 macroconidia × 650

Recent infections may be undetectable at harvest, and consequently infected bulbs rot in store (4,8); however, there is no evidence of spread from bulb to bulb.

CONTROL The fungus may be transmitted in seed or sets (1,3,6), necessitating fungicide treatment before planting (6). Soil disinfestation can be achieved by solarization (page 132). In some localities it may be beneficial to remove certain weed species (*Oxalis* spp.) on which the fungus can thrive in the absence of the bulb crop. The best long-term strategy, however, is to breed new cultivars with resistance or tolerance to the disease, and this is being done for both onion (4) and garlic (9).

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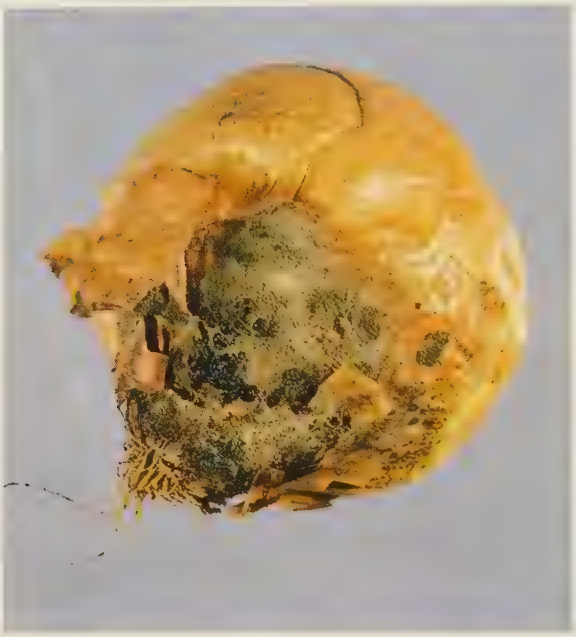
GREY MOULD ROT and NECK ROT of onions, shallots and garlic caused by

***Botryotinia* spp.**
Conidial state: *Botrytis* spp.

322



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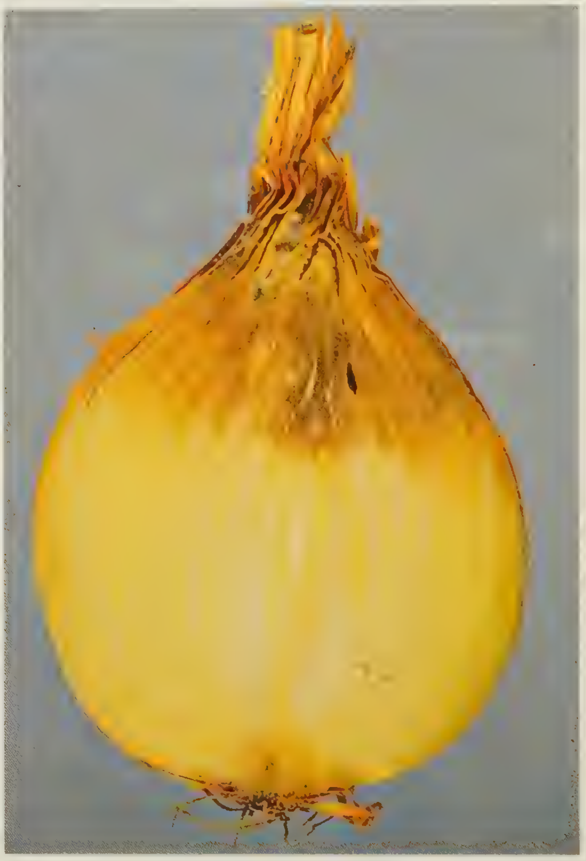
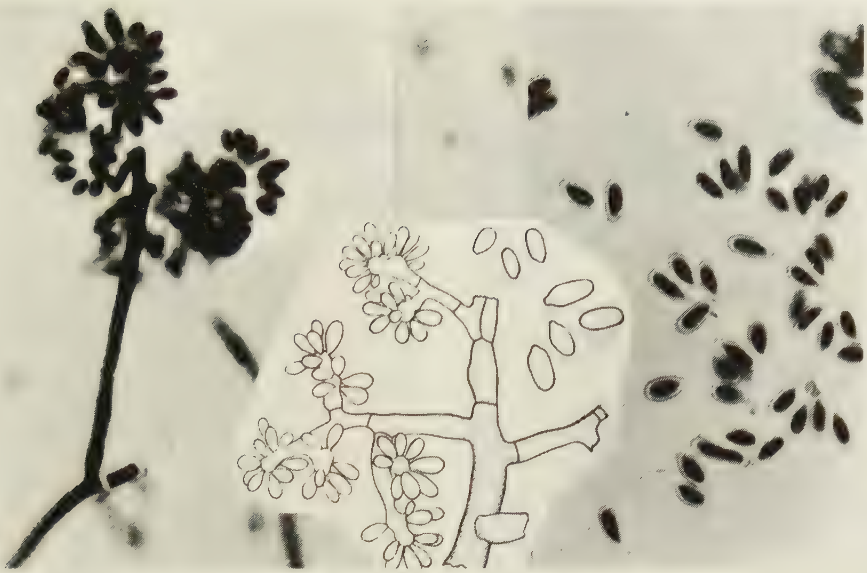


OCCURRENCE *Botrytis allii* Munn is the most important of the three species of *Botrytis* which cause neck rot of onion bulbs (10). The other two are *B. byssoidea* Walker causing mycelial neck rot, and *B. squamosa* Walker, causing small sclerotial neck rot. *B. porri* Buchw. is the cause of dry rot of garlic, reported from Hungary (7) and the USA (6). The following account relates primarily to *B. allii*, on which most of the work has been done. This species appears to exist only in the asexual state (conidia). The disease is particularly important in temperate countries and in subtropical regions having a cool wet season (CMI Map 169); it has been the subject of investigation in the UK (4,5), the Netherlands (8), Germany (2), Israel (3), Egypt (1) and the USA (10).

SYMPTOMS The fungus may be present in any part of the onion bulb but it is characteristically found in the neck region, from where it spreads downwards into the rest of the bulb. Infected tissue is soft and discoloured, with a clearly defined margin. A mass of grey powdery spores develops, which may later be accompanied by black bodies (sclerotia) 1–5 mm in diameter. *B. byssoidea* produces similar symptoms, except that spores are rarely formed (10), while *B. squamosa* is characterised by the production of flat scale-like sclerotia (10).

BIOLOGY A major source of this disease, at least in Europe, is infected onion seed, which may be carrying the fungus in its internal tissues (2,5). Such seeds give rise to infected seedlings but, since no symptoms are produced at this stage, healthy and infected plants are at first indistinguishable in the field. During the growing season the leaves are further invaded and, under humid conditions, spores (conidia) are produced on the dying tissue. Frequent periods of rain favour release of the spores and infection of healthy plants; thus in wet seasons even low levels of seed infection can have serious consequences. The fungus grows down the leaves and may be deep within the neck tissues of the onion bulb by the time it is harvested. An infected crop may be in apparently good condition when shipped or put into store, though if sample bulbs are incubated under controlled conditions the latent fungus can be detected by a simple staining technique (3). During transport and storage infected bulbs undergo progressive decay at a rate determined by the



324 *Botrytis allii* (from CMI Descr. No. 433, Ellis & Waller 1974) Conidia and conidiophores × 500

ambient temperature. Contrary to general belief, however, the disease does not spread into adjacent healthy bulbs; the percentage of rotted bulbs in store is a direct reflection of the percentage of plants with neck infection in the field.

CONTROL For control of grey mould neck rot, emphasis has long been placed on efficient drying or curing of the onions after harvest (1,4). Bulbs should be removed from the field within 48 hours of topping, to avoid infection of the damaged green neck tissue (4). Thorough drying (at 30°C) can check the advance of the fungus and prevent its becoming established in the fleshy scales. The primary means of control, however, is to sow healthy seed (5); commercial onion seed should be treated with a systemic fungicide before being sold to growers. Since *Botrytis* may be carried over from one season to the next in onion debris in the field, it is also wise to practise crop rotation if neck rot has been a problem. Work is in progress to breed resistant varieties of onion (9).

---

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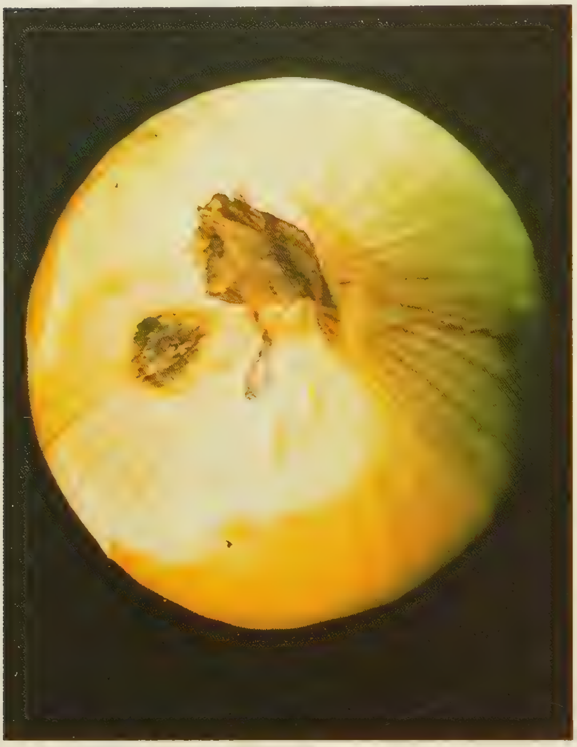
PURPLE BLOTCH of onions, shallots and garlic caused by *Alternaria porri* (Ell.) Cif.

325



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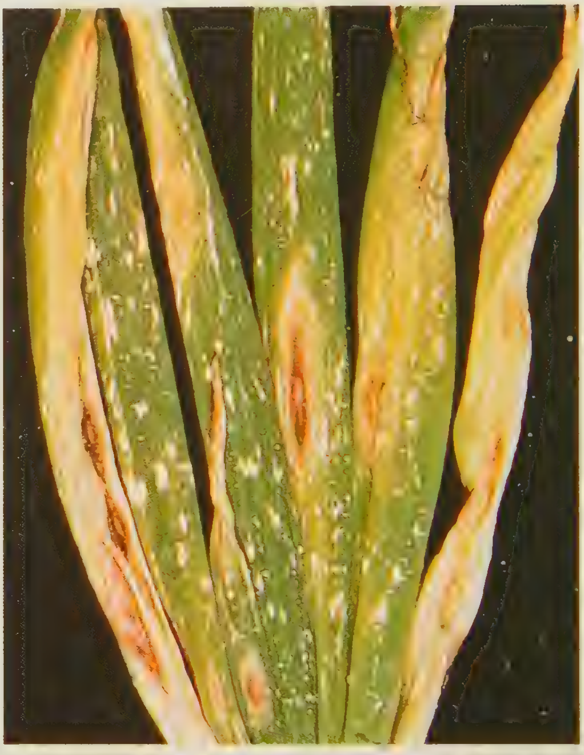
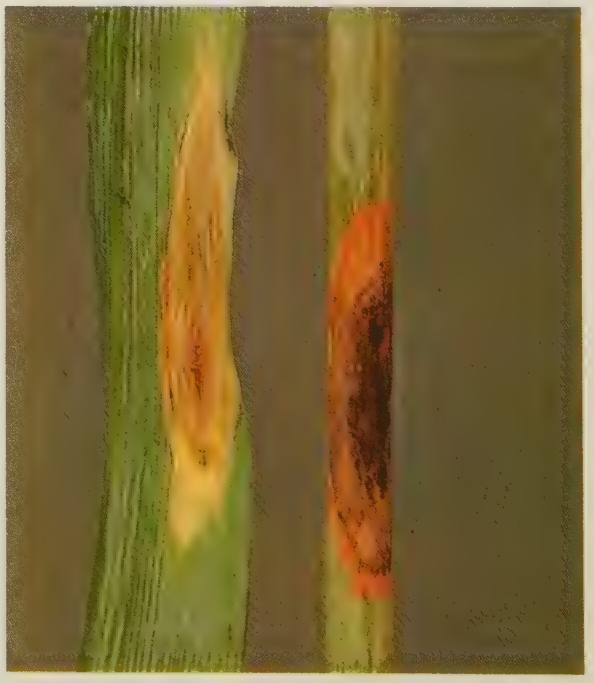


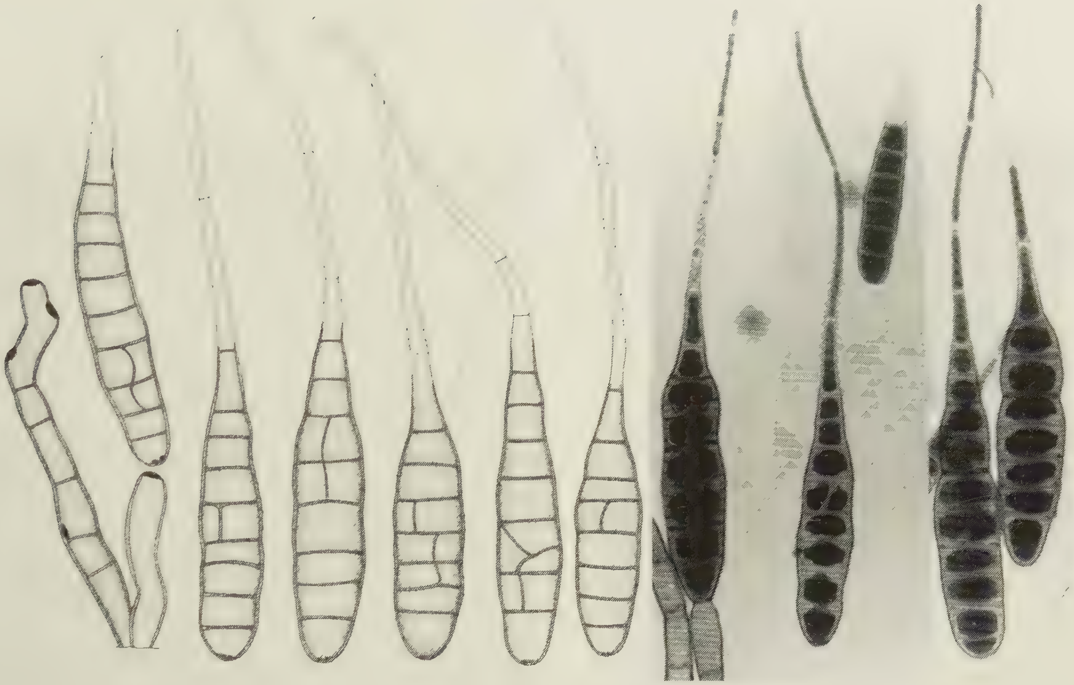
OCCURRENCE This disease is widespread, and severe losses have been recorded on onions in India (1,6,10), Kenya, Egypt (5), Cuba (3), Puerto Rico, and many parts of the USA (2,7,9). Records on garlic include India (8) and Thailand (4).

SYMPTOMS Rotting of bulbs usually begins in the neck region, but it may also start at wound sites. The decay is semi-watery, and characteristic pigments are produced in affected scales, firstly yellowish, later deepening to reddish-purple and finally almost black (2). The purple stains should not be confused with those produced by a certain species of blue mould (q.v.).

BIOLOGY The purple blotch fungus survives in the soil on crop debris, and new infections occur in the field. The formation of asexual spores (conidia) is favoured by rain or irrigation, coupled with temperatures between 20° and 30°C. The spores are dispersed by air currents, and infection can occur by direct penetration of healthy leaves (5), though insect attack renders plants especially vulnerable to fungal invasion (10). Small greyish-white lesions are produced on the leaves and, if conditions remain moist, may develop into elliptical purplish areas several cm in length with light and dark zonation and masses of brownish-black spores (2). The bulbs become infected when they are 'topped' at harvest-time, and later undergo rotting in store (9).

327





328 *Alternaria porri* (from CMI Descr. No. 248, Ellis & Holliday 1970) Conidia and conidiophores × 500

CONTROL Purple blotch severity is influenced by cultural practices such as density of planting and fertiliser applications, and can be reduced by summer ploughing and early sowing (6). Fungicide sprays can be applied (1,4) but the disease becomes increasingly difficult to control as the crop approaches maturity (7). Some cultivars show resistance to the disease; in garlic it was noted that cultivars possessing curved drooping leaves were more susceptible than those with flat erect leaves (8).

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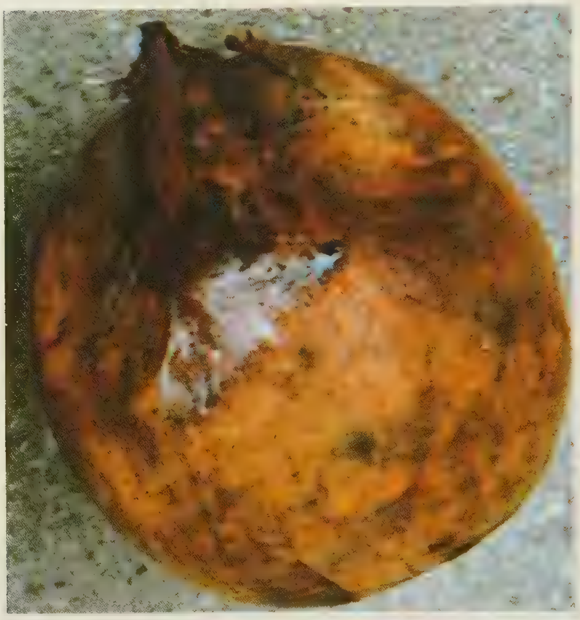
SCLEROTIUM ROT of onions, shallots and garlic caused by

Corticium rolfsii Curzi

Sclerotial state: *Sclerotium rolfsii* Sacc.

329



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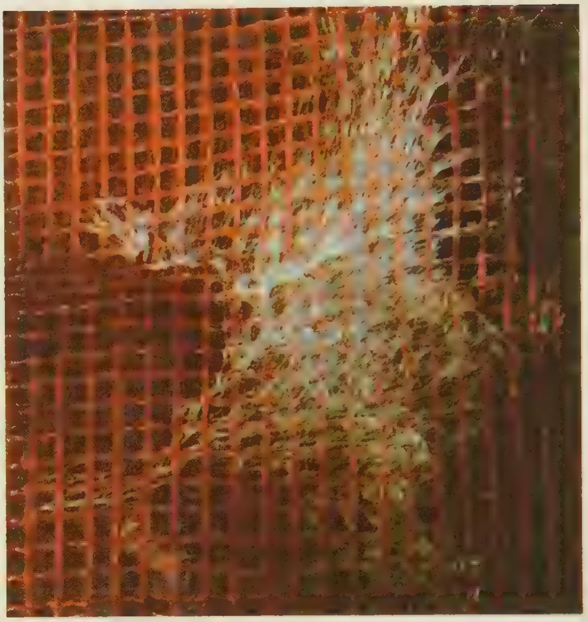

OCCURRENCE This fungus is widespread in the moist tropics and warm temperate regions, and has been recorded on onions in India (5,9), Egypt (6), Israel (3), Australia (7) and the southern states of the USA (4,10). The writer has observed it on onions imported into the UK from New Zealand. Shallots are attacked in the USA, and garlic in the USA (2) and Japan (1).

SYMPTOMS Initially, dirty white spots may be seen on the outer scales; eventually the entire bulb becomes soft and rotten (5). A bright white silky mould, characteristically fan-shaped, spreads over the surface and also tends to grow on the nets in which the produce may be packed. Small spherical bodies (sclerotia) develop on the mould, mostly 1–2 millimetres in diameter (cf. white rot, q.v.) when fresh and pale, later shrinking slightly and becoming hard, dark brown and dry (2,4).

BIOLOGY The fungus exists in the soil in the form of sclerotia (2), and also colonises vegetative debris (8); the sexual state (involving the formation of basidiospores) has been recorded but is not important in infection. Under warm moist conditions in the field, fungal strands grow out from sclerotia near the soil surface and are capable of direct penetration of the developing plant (4). The fungus grows rapidly at temperatures between 25° and 35°C, and proliferates after wet weather or irrigation (3). The crop may rot in the field; alternatively recent infections may escape detection at harvest but cause losses in store. Under refrigeration, however, fungal growth is completely checked.

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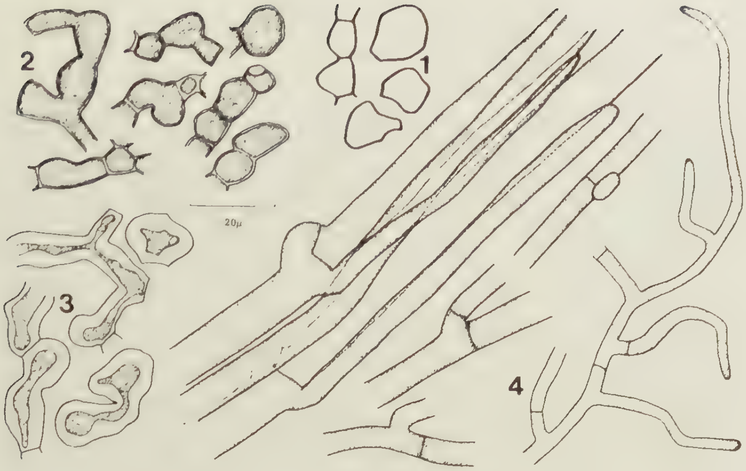

332 *Corticium rolfsii* (from CMI Descr. No. 410, Mordue 1974) 1 cells from sclerotium rind 2 cells from sclerotium cortex 3 cells from sclerotium medulla 4 hyphae

CONTROL The disease, which is known as southern blight (8), may be controlled in several ways, according to locality. An integrated approach is required, which may include biological control (8). With regard to crop rotation, cereals and grasses are among the few crops unaffected by this fungus (2,4,8). Cultural practices such as deep ploughing can be helpful in burying crop debris infested with sclerotia, before the new crop is planted (4,8). Alternatively the sclerotia may be killed with a soil fumigant or by solarization (page 132) (8). Weed control is important (8). The harvested crop may be treated with a fungicide, in the form of a dust rather than a dip (7). An observer in the USA noted that, in comparison with a white cultivar, a coloured cultivar of onion seemed to be highly resistant to the disease in the field (10). Because of its high temperature requirement the fungus is unknown in the UK but, in view of its wide host range and because of fears that it could become established in heated glasshouse crops, it is a notifiable disease organism and strict quarantine measures are enforced if it is intercepted on imported produce.

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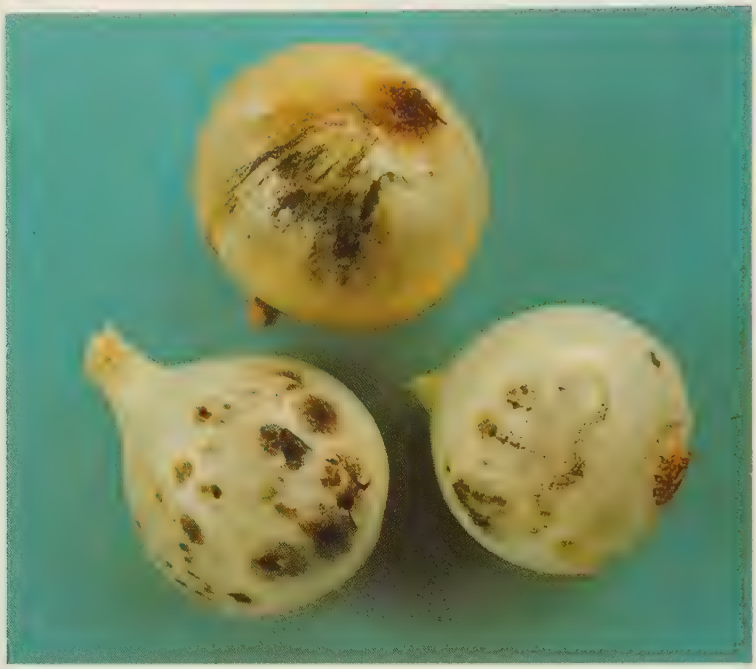
SMUDGE of onions, shallots and garlic caused by ***Colletotrichum circinans*** **(Berk.) Vogl.**

333



OCCURRENCE Smudge is widespread in the USA (10), Europe (1) and Japan (5). It is generally found only on white cultivars (4), on which it causes an unsightly blemish. Occasionally, however, the disease affects coloured onions (2).

SYMPTOMS The external symptoms are minute dark green to black bodies, often in concentric circles, forming smudgy spots of the order of 1 cm in diameter (9). Under moist conditions these minute bodies develop into asexual spore-bearing structures (acervuli), in which cream-coloured spore masses and dark hair-like projections (setae) may be seen with the aid of a hand lens (5,9). Infection of underlying scales may lead to the formation of yellowish spots.

BIOLOGY The fungus is transmitted to new areas on infected planting material and can persist in the soil for many years. Warm wet weather provides favourable conditions for infection of a susceptible crop, and the spores (conidia) are disseminated by rainsplash (10). The fungus is capable of direct penetration, without the necessity for wounding (7,8). In addition to disfiguring the bulbs, it also induces premature sprouting (9).

CONTROL The first observers of onion smudge noticed that, in contrast to white varieties, coloured cultivars usually remained free of the disease when growing in contaminated soil. It was later demonstrated that natural substances in the outer scales of coloured cultivars prevent normal development of the fungus (2,6). The main strategy for avoiding losses due to smudge is therefore to grow coloured cultivars possessing well-developed outer scales (10). If white cultivars are grown in infested areas the bulbs should be well dried (cured) immediately after harvest.

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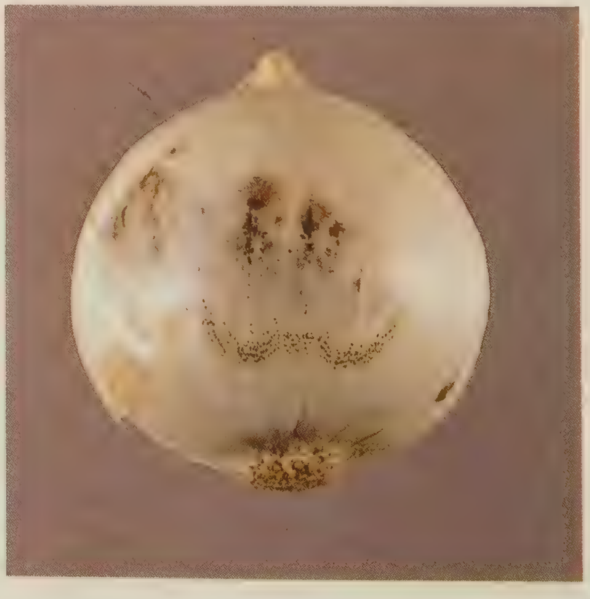
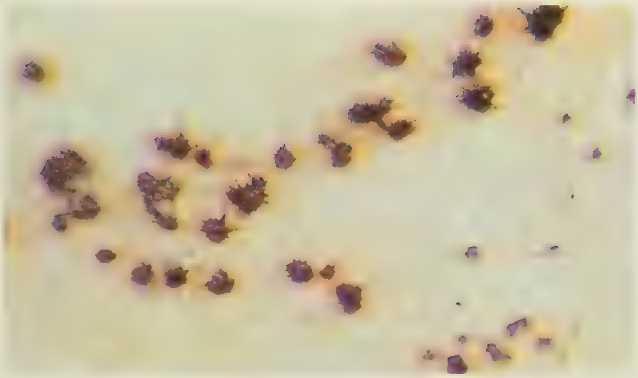
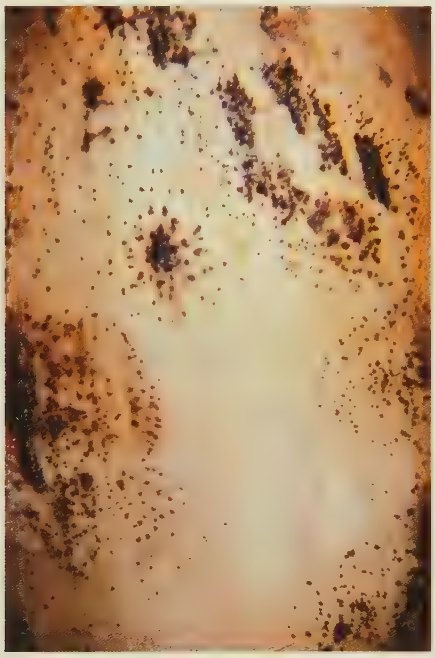




335 Close-up of acervuli, showing setae



336 Acervuli on onion

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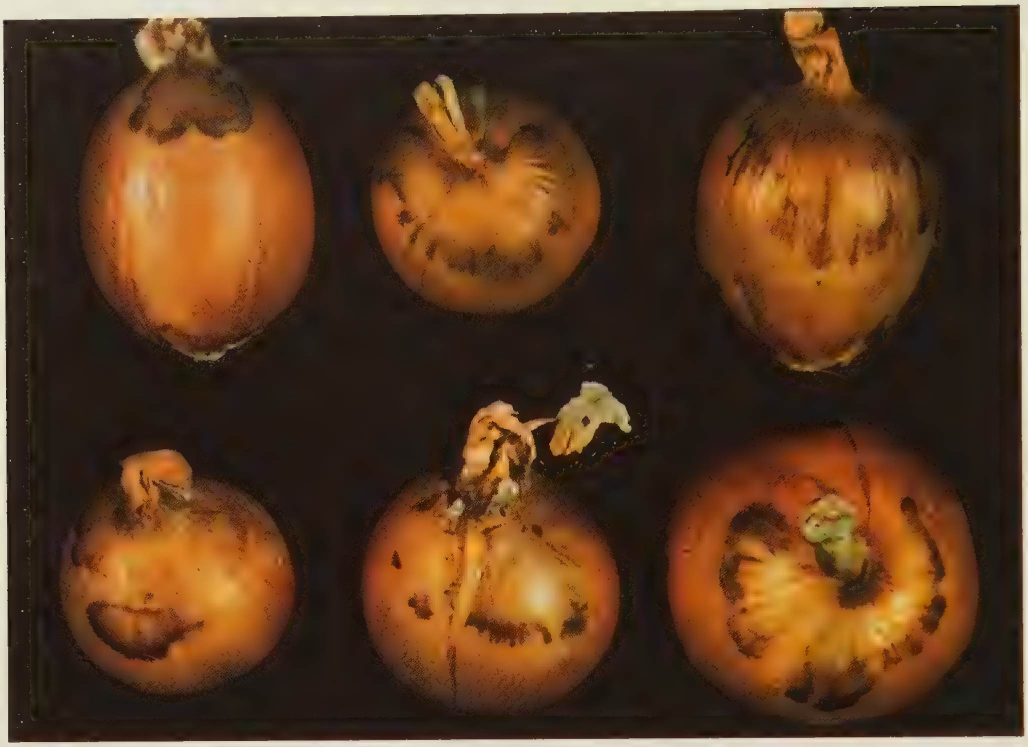
OTHER DISEASES of onions, shallots and garlic



337 Brown stain of onions

BOTRYODIPLODIA ROT caused by *Botrodiplodia theobromae* (page 371) has been recorded on Texas-grown white onions; it does not attack coloured cultivars. The fungus causes a silvery-grey blemish on the neck and shoulder, and a dry leathery decay (7).

BROWN STAIN of onions, induced by *Botrytis cinerea* (page 281) is characterised by a dark-edged discoloration of the outer scales, extending from the neck to the shoulder, or circular spots in any position on the exterior of the bulb (1). Black resting bodies (sclerotia) 1–3 mm in diameter sometimes form at the centre of the spots, and the main effect is one of disfigurement, the interior of the bulb being unaffected. In the USA red and yellow cultivars were found to be susceptible while white cultivars showed resistance (1).

MACROPHOMINA ROT has been recorded on onions in Australia and on onions and garlic in India (3). The fungus, *Macrophomina phaseolina* (page 25), causes a hard brown rot, and the disease may be controlled by post-harvest fungicide treatment.

PINK ROOT is a serious field disease of onions and garlic, for example in Israel and the USA (4). The causal fungus is *Pyrenochaeta terrestris* (Hansen) Gorena, Walker & Larson. Healthy roots may be attacked at all stages of development and, although the pink root fungus does not progress into the bulb, it often opens the way for *Fusarium oxysporum* f.sp. *cepae*, the cause of basal rot (q.v.). In regions having a hot season, both fungi may be controlled by solarization (page 132). Some cultivars show marked tolerance to pink root disease.

RHIZOPUS ROT of garlic has been reported from India, the causal fungus being *Rhizopus oryzae* (page 373). A coarse white mould bearing black spore-heads (sporangia) develops on the cloves; the disease may be controlled by post-harvest fungicide treatment (8).

RUST, caused by *Puccinia allii*, is sometimes a limiting factor in the production of garlic, and can also affect onion. Being essentially a leaf disease, it is considered in detail under Diseases of Spring Onions and Leeks (page 226).

SCLEROTINIA ROT, caused by *Sclerotinia sclerotiorum* (page 47), has been recorded on stored onions in the USA and on garlic in Taiwan (10). A white mould develops, on which form black resting bodies (sclerotia).

SMUT, caused by *Urocystis cepulae*, is essentially a field disease on onions, and is described under Diseases of Spring Onions and Leeks (page 228).

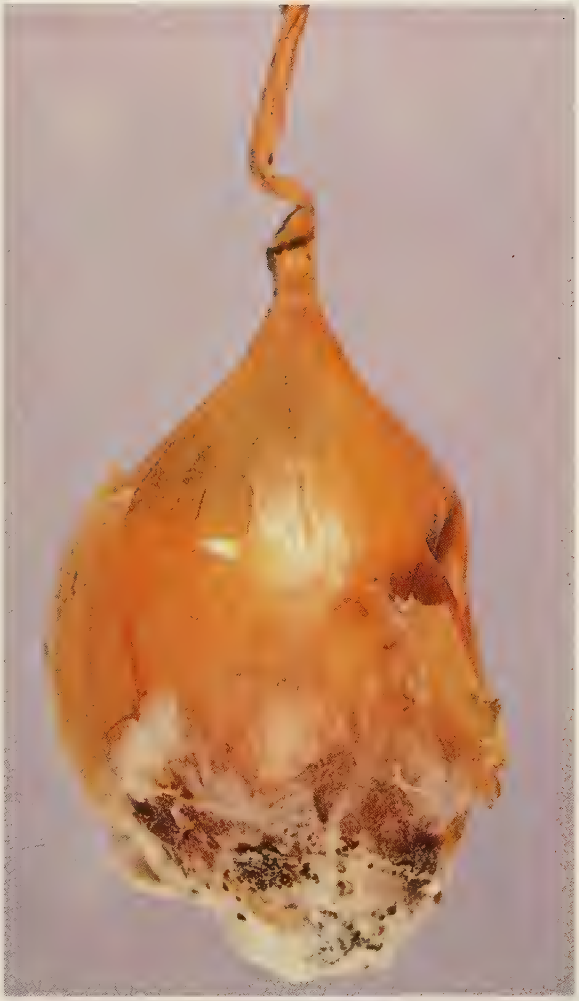
SOUR ROT is caused by *Geotrichum candidum* (page 288) and the disease has been recorded in the USA (6). In susceptible onion cultivars translucent watery lesions develop rapidly at temperatures between 20° and 30°C.

WHITE ROT, caused by *Sclerotium cepivorum*, is becoming increasingly important on onions, shallots and garlic (2,9); details are to be found under Diseases of Spring Onions and Leeks (page 230).

WHITE TIP and bulb rot, caused by *Phytophthora porri*, are described under Diseases of Spring Onions and Leeks (page 232).

YEASTY ROT was earlier confused with bacterial soft rot (q.v.) but is now known to be caused by the yeast *Kluyveromyces marxianus* (Hansen) van der Walt var. *marxianus* (5). The soft watery rot tends to be confined to the inner or outer fleshy scales, and does not spread easily from scale to scale. At temperatures between 20° and 30°C, however, rotting is rapid and much of the bulb may be affected.

YELLOW MOULD ROT is caused by *Aspergillus alliaceus* Thom & Church, and has been recorded on garlic from Italy. Small black resting bodies (sclerotia) develop in the decayed tissue, and under high humidity a mass of yellow spores forms on the cloves. The optimal growth temperature is above 30°C, and fungal growth is inhibited at temperatures below 20°C.



338 White rot of onion

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SPROUTING and **ROOTING** of onions, shallots and garlic

OCCURRENCE These are natural processes but, being undesirable in bulbs intended for consumption, are classed as 'market disorders'. Sprouting and rooting can occur independently of one another (5,6).

SYMPTOMS Incipient sprouting is indicated by the presence of yellowish-green leaf initials in the neck of the bulb, visible if the bulb is sectioned (2). At a later stage the shoot emerges from the neck and can reach a length of over 30 cm. The first sign of root formation is a swelling of the basal plate, sometimes followed by cracking of the papery scales in this region. A horizontal cut through the basal plate shows the root initials as a ring of brownish 'dots' (2). When they emerge, new roots are white and plump, about 1 mm in diameter, in contrast to the remains of old roots which are dark and withered.

BIOLOGY It used to be thought that, after harvest, onions entered a period of 'rest', during which there was almost complete inactivity regardless of environmental conditions. It is true that dormant bulbs have a very low rate of respiration and appear to change little, but it is now known that within the bulb there is a continuous process of growth, very slow at first but gradually accelerating (1). Sprouting is the eventual result, and the accompanying rise in respiration rate leads to substantial heat output. The rate at which dormancy is lost depends on several factors. Cultivars vary greatly in their tendency to sprout; for example in the cultivar Excel it was found that (at 17°C) 50% of the bulbs had sprouted only 40 days after harvest, while in cv. Rijnsburger over 200 days elapsed before sprouting reached a comparable level (1). The cultivar grown in Chile, cv. Valenciana, is intermediate between these two. It is a general rule that mild varieties sprout more readily than pungent types. Also influential are season, site, and maturity at harvest; in one study it was found that in summer crops early-harvested bulbs sprouted soonest while in winter crops it was late-harvested bulbs which sprouted soonest (4). Bulbs with internal disease tend to sprout prematurely. Temperatures between 10° and 20°C are most conducive to sprouting (1). Below 10°C all biological processes are slowed down, while at 25°C and above, sprouting is inhibited (1). Atmospheric humidity seems to exert little effect on sprouting, but is of prime significance in stimulating root formation (5). If the relative humidity is greater than about 85% then the basal plate becomes damp and roots are initiated within a few days. This can occur at any temperature.

CONTROL An effective and long-established method of inhibiting sprouting is to spray the crop with the growth-regulating chemical maleic hydrazide (2,4). The treatment should be applied about 2 weeks before harvest while the tops are still green, so as to ensure translocation of the active ingredient from leaves to bulb. It is essential to harvest the crop at the correct maturity and to subject the bulbs to a proper curing process. High temperature storage (25° to 30°C) delays sprouting but favours decay (1). Low temperature storage (at around 0°C) inhibits both but is expensive, the more so because of the additional necessity to maintain a relative humidity below 85% for prevention of root growth. During marine carriage good ventilation is essential in cargoes shipped at ambient temperature, while in refrigerated space it is desirable to take special measures to maintain relative humidity as low as possible (page 238). If legislation permits, harvested bulbs may be subjected to irradiation treatment, which inhibits both sprouting and rooting (3).

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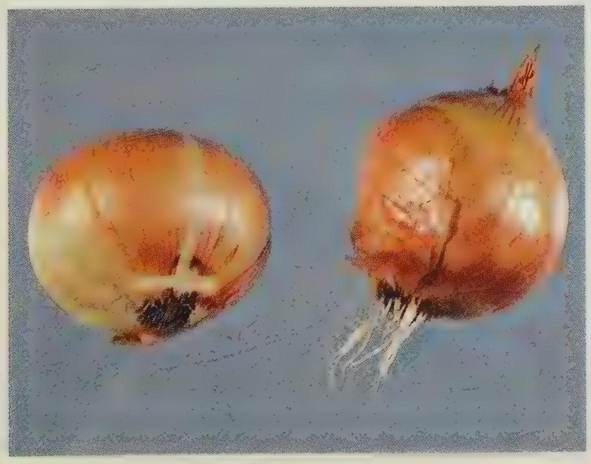
OTHER DISORDERS of onions, shallots and garlic

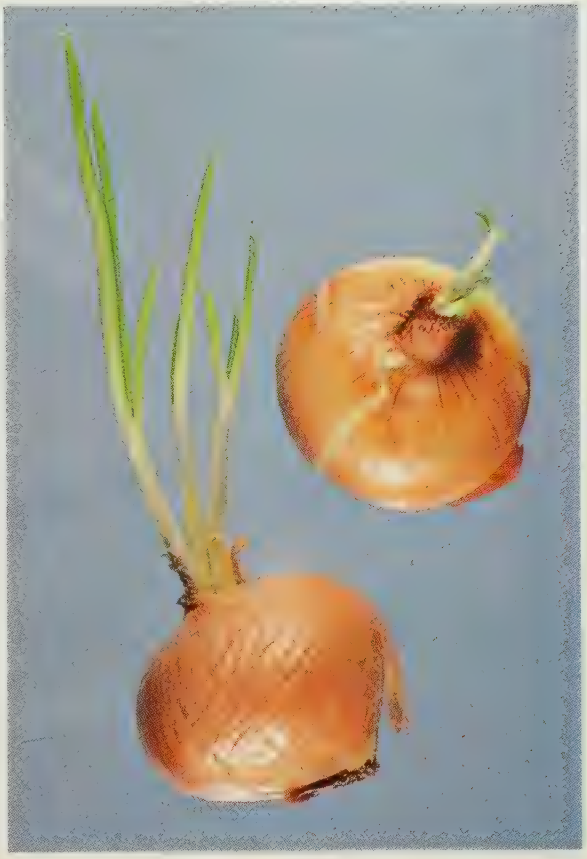
BRUISING of onions tends to result in watersoaking of the outermost fleshy scale(s), and it is sometimes difficult to distinguish the symptoms from those of freezing injury (q.v.) and translucent scale (q.v.). Observations of the pattern of damage within a consignment can assist in diagnosis; for example, it may be noticed that damage is especially severe in those parts of the load located over the rear axle of a vehicle, suggesting bruising during transit (5). Re-examination in subsequent weeks may show a much reduced incidence of injury, since even severely bruised bulbs have a capacity for recovery (5).

FREEZING INJURY of onions may affect the outermost fleshy scales or the entire bulb, according to the duration of freezing temperatures. Thawed onion tissue appears watersoaked in cross-section but, in contrast to 'translucent scale' (q.v.), the freshly cut surface tends to appear dry. A further distinguishing feature is the tendency for the scale's covering membrane (epidermis) to separate from the scale. Onions freeze at approximately −1°C, the exact temperature depending on cultivar and soluble solids content. Supercooling may occur, however, (page 334) and injury may be avoided if warming up can be achieved without vibration (3).

'SKINNING' is a colloquial term used to describe the cracking and subsequent loss of outer scales (skins), sometimes to the extent of leaving the fleshy scales exposed. Skin-cracking may be caused either by mechanical shocks to the bulbs, or by changes in bulb shape associated with re-growth. Brittleness of skins is determined partly by inherent physical properties such as scale-thickness, which varies with cultivar. Also influential, however, are environmental factors affecting moisture content, since if the skins contain less than 20% moisture they are notably brittle. Considerable expertise may be involved in manipulating temperature and humidity during curing and storage; if drying is too rapid or too thorough, there is an increased risk of cracking and skin-loss during subsequent handling. Before onions are graded out of cold store, it may be advisable to 'condition' the bulbs with warm, slightly moist air which allows the skins to remain flexible. Care is necessary, however, to prevent condensation (Currah & Proctor, 1990).

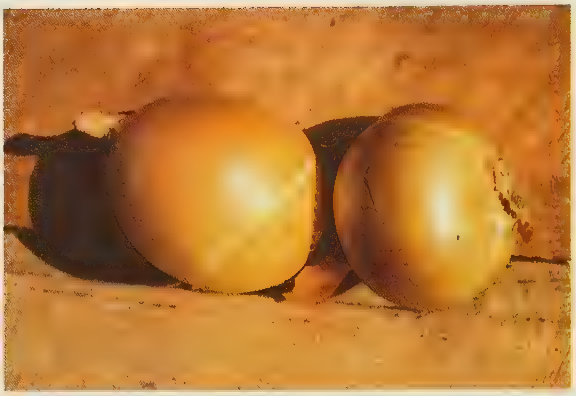


339 Root-formation



340 Sprouting

THICK-NECKING of onions refers to a failure to develop mature bulbs in the field (1). Onions with thick necks are difficult to dry after harvest, and are predisposed to sprouting and decay during storage. Incomplete bulbing may be the result of delayed sowing, a cool growing period, or the over-use of fertiliser. Cultural practices which promote early maturity can therefore be advantageous.

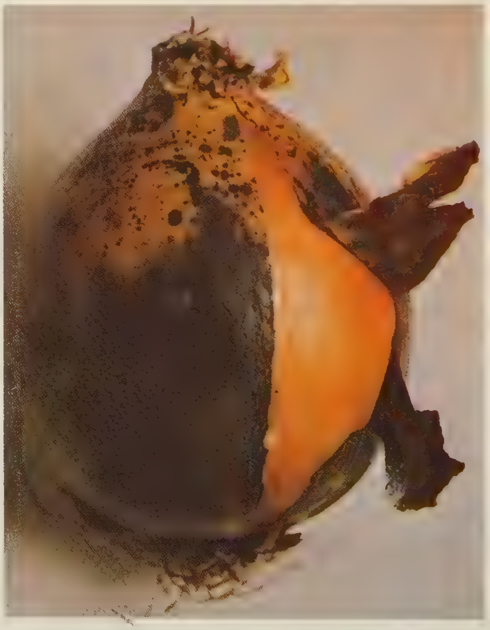


341 Skin-splitting

TRANSLUCENT SCALE of onions refers to a clearing and watersoaking of normally opaque cells which, on microscopic examination, are seen to be completely disorganised (2). The disorder tends to affect the second or third fleshy scale (cf. freezing injury, q.v.) and symptoms develop only after a period of storage. Predisposing factors are thought to include high temperature during the latter part of the growing season, and delay between curing and cooling for refrigerated storage. In a hot climate it may be advisable to cover maturing bulbs with soil.

WATER-STAINING of the outer scales is characterised by discoloration ranging from dull grey blotches to a deep blackness resembling scorching by fire (4). The paler type of staining may be associated with superficial mould growth following wetting of the bulbs by rain or by condensation. A more intense discoloration can be induced by sea-water. Staining also results from the action of alkaline substances which, under damp conditions, may diffuse out of certain fabrics used to make bags; the characteristic pattern on the onion skin is a dark imprint of the mesh known as 'bag-print' (4). Staining is sometimes traced to wet conditions in the field during and after harvest; the problem can be minimised by harvesting the crop directly and curing it in store instead of in the field. Good ventilation is necessary to dissipate respiratory moisture during storage and transport. In marine carriage especial care must be taken to prevent ingress of sea-water.

WAXY BREAKDOWN of garlic consists of a yellowish discoloration in part or all of the flesh of individual cloves. There may be no external indication of breakdown until the advanced stage is reached, when the clove has become shrunken and a dark amber colour is visible through the outer papery scales. Little appears to have been published on the disorder, except the observation that it tends to be associated with excessively high temperatures around harvest time. Occasionally consignments of garlic suffer over-heating as a result of poor ventilation during shipment; in this case, entire heads may suffer physiological breakdown.



342 Sea-water staining

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See also:

CURRAH L. & PROCTOR F.J. (1990) Onions in tropical regions. *Bulletin of the Natural Resources Institute* No. 35, 232 pp illus.

CHAPTER 8

TEMPERATE ROOTS AND TUBERS

True root vegetables include carrots and parsnips. Potatoes are grown for their tubers (swollen underground stems) while the edible part of beets, radishes, swedes and turnips consists of both root and stem tissue, loosely referred to as 'roots'.

Beets

Beets belong to the family Chenopodiaceae, and *Beta vulgaris* L. encompasses red beet, sugar beet, fodder beet and chard (q.v.). Primitive beets have long been grown in Europe, but it was not until the 18th century that selection was undertaken for high sugar content; sugar beet now accounts for more than half of the world's sugar. The most important of the vegetable beets is red beet, otherwise known as garden beet, table beet, or beetroot. The roots, which may be round or long, are boiled and then peeled, this procedure being necessary to avoid leakage of crimson juice. For the same reason, beetroots need to be harvested with especial care if damage, with consequent 'bleeding', is to be avoided. Red beets are especially popular in northern and central Europe, the USSR, the northern USA and Canada. There is also a trade between Cyprus and the UK.

Beets can be marketed in bunches with the foliage still intact, and in this state may be held for one or two weeks at 0°C. Crushed ice is beneficial. More usually beets are topped (preferably not too close) in preparation for storage over a period of months. There are three main hazards: first is moisture loss, which causes beetroots (especially small ones) to become flaccid and even shrivelled; second is shoot-growth, and third is decay. The last two are sometimes linked, in that new shoots may die and initiate deterioration of the root; this is especially likely in the context of boron deficiency, which is also responsible for the disorder 'internal black spot' (3,5,9). Decay may also be caused by a complex of fungi, against which pre-harvest control measures are necessary. To minimise physiological and pathological deterioration, high humidity and low temperature are essential; a temperature of 0°C is often quoted, though other studies have demonstrated that 4°C is preferable. For shipment of beetroots, refrigeration is necessary and it is appropriate to pack beets in polyethylene-lined sacks (with perforations). The liner must envelop all the beets since, if some remain uncovered, these will lose moisture preferentially and suffer extreme wilting.

343 Moisture loss in beets due to faulty packaging

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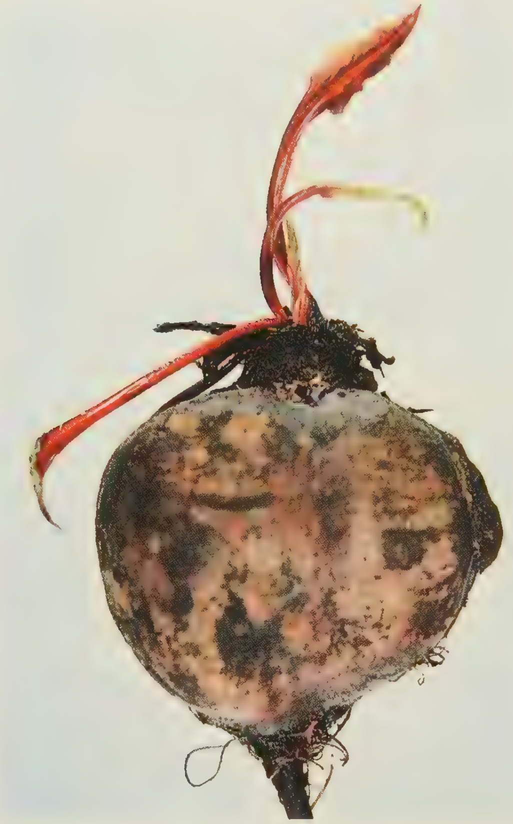
PHOMA ROT of beets caused by

Pleospora betae **(Berl.) Nevodovsky**
Conidial state: *Phoma betae* Frank

344



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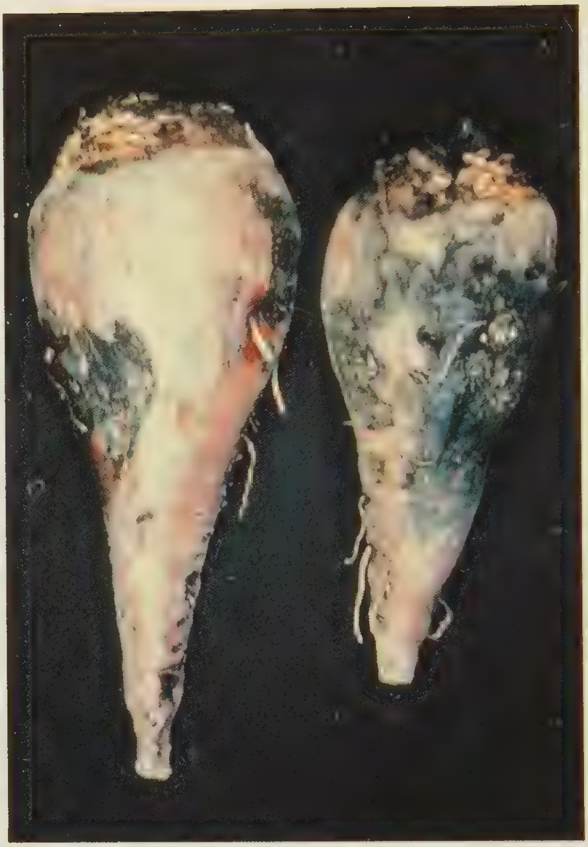
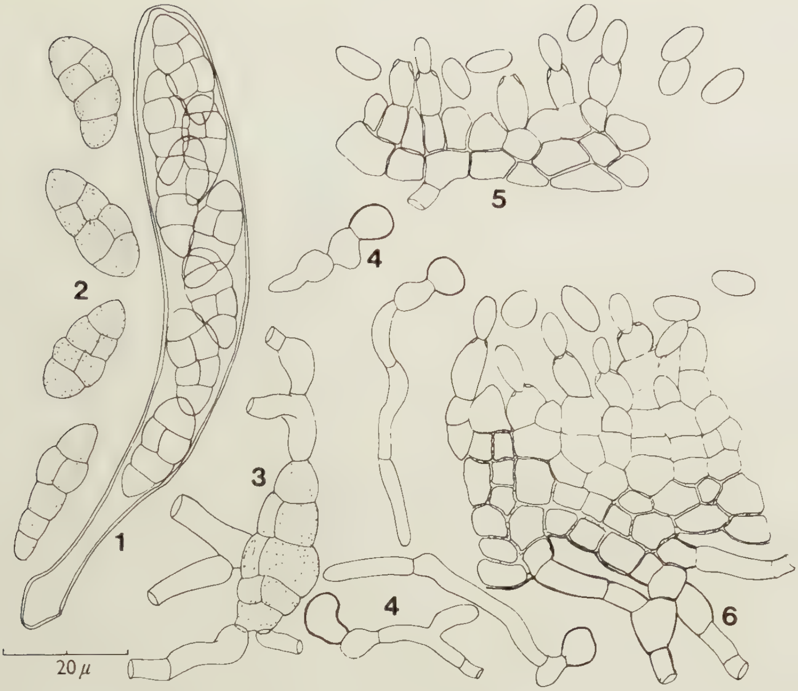


OCCURRENCE The fungus is common in temperate countries, including the UK (3,5,10), Sweden (1), Italy (7), Canada (4) and the USA (2,6,8,9). Much of the work done on this disease relates to sugar beet (1,2,3,4,6), but table beets have also received attention (8,9,10).

SYMPTOMS Early lesions in table beets are not easily discernible because of the dark red background colour. Decay may begin at the root tip, at the crown or in wounds. Affected tissue turns black and is slightly depressed; later the surface becomes covered with a greyish-white mould (8). Internally the decayed areas are at first brown and water-soaked, later black and granular, with a sharp line of demarcation between diseased and healthy tissue. Eventually the tissue dries out and becomes spongy, containing cavities lined with mould (8).

BIOLOGY The fungus is seed-borne, and seedlings may develop 'blackleg' (3,10). Under conditions favourable to plant growth, however, many seedlings survive the attack but continue to harbour the mould in a dormant state (6). If, at a later stage, plant vitality is impaired, the fungus becomes active again and invades the vascular system of both stems and roots (6). Black fissures are characteristic external symptoms on roots (2). Infection may also occur through wounds at harvest time, from spores in the soil (2). Roots appearing healthy may later undergo severe decay in store (8), especially if they have been allowed to wilt (4). In the field the fungus survives the winter in beet debris, and the sexual stage (perithecia) has been recorded on dried stems, including those of wild beet (5). During the summer ascospores are liberated; they are carried by wind and infect foliage of the growing crop (1). Leaf-spotting is especially common in second-year beets grown for seed (1). The asexual state (pycnidia giving rise to conidia) develops in leaf and stem lesions, and these spores are dispersed by rain. Contamination of seed may occur before or during threshing operations; wet conditions around harvest time can lead to deep-seated infection (1), with pycnidia developing on the seed-coat (3).



346 *Pleospora björlingii* (from CMI Descr. No. 149, Booth 1967) 1 ascus 2 ascospores 3 germinating ascospore 4 germinating conidia 5, 6 part of pycnidial wall

CONTROL Field hygiene is important, and removal and destruction of beet debris is strongly advisable (1). It is essential to treat beet seed with a fungicide (10), though this may not give complete control if the soil is infested (2). Seedlings may be protected by the use of a soil amendment containing an organism antagonistic to the fungus (9), and fungicide sprays have also been advocated for the growing crop (7). Harvested beets should be promptly stored before they can undergo wilting (4).

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See also:

MONTE E., MARTÍN P.M. & GARCÍA-ACHA I. (1989) Pycnidial development in *Phoma betae. Mycological Research* **92,** 369–372 illus.

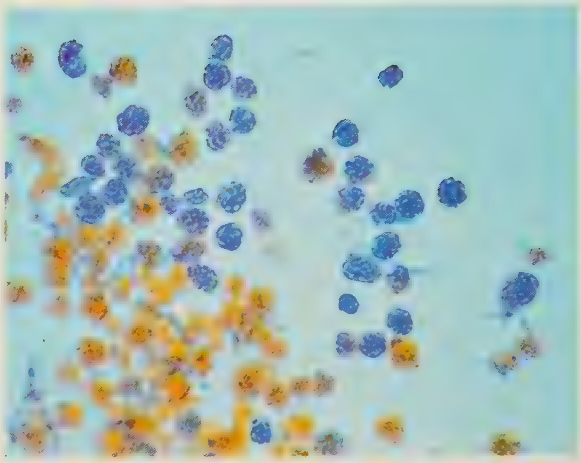
OTHER DISEASES of beets

BACTERIAL SOFT ROT may be caused by *Erwinia* spp. The bacteria are soil-borne, and can attack the growing crop, invading the vascular system and causing a root rot (9). Infection by soft rot bacteria can also occur after harvest, via injured foliage. Decayed tissue is soft, moist and evil-smelling.

PYTHIUM ROT may be caused by *Pythium ultimum* (page 328), usually associated with 'damping off'. If infected plants survive the seedling stage, the fungus may later colonise the fleshy roots. In the USA trials have been carried out in which an antagonistic fungus is added to the soil to reduce the population of *Pythium* (3) (see also ref. 1 on page 263).

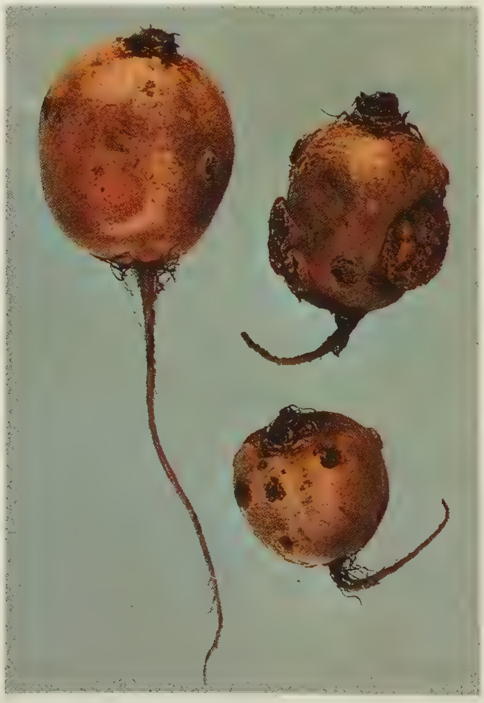
RHIZOCTONIA ROT is caused by *Rhizoctonia solani* (page 306). In the USSR this disease (known as 'brown rot') is controlled by means of crop rotation with maize and grasses, and by the use of mineral fertilisers rather than manure (5).

RHIZOPUS ROT of sugar beet has been recorded in Canada, where *Rhizopus oryzae* (page 373) was found to be causing root rot in the field (2,8). It is conceivable, therefore, that table beet might be subject to the same disease.

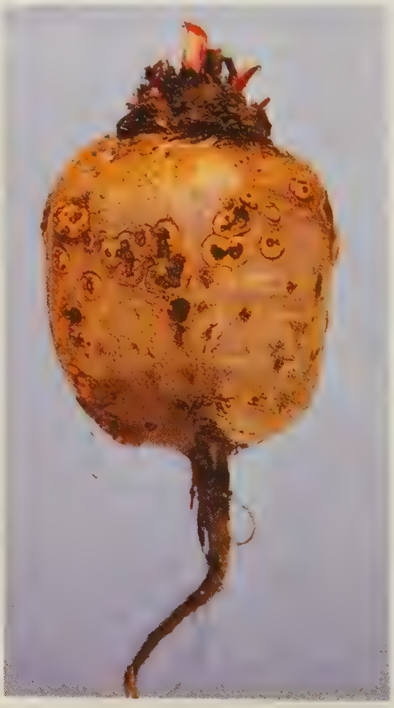


348 Spore-balls of *Spongospora subterranea* (stained with cotton blue in lactophenol) (see page 320)

SCAB is caused by *Streptomyces scabies*, the same organism which causes common scab in potatoes (q.v.). Lesions are protuberant and disfiguring. Beet plants are susceptible only from the fourth to the sixth week after sowing (after decortication and before suberization of the roots), and so short-term application of a protective chemical should give sufficient control (1).



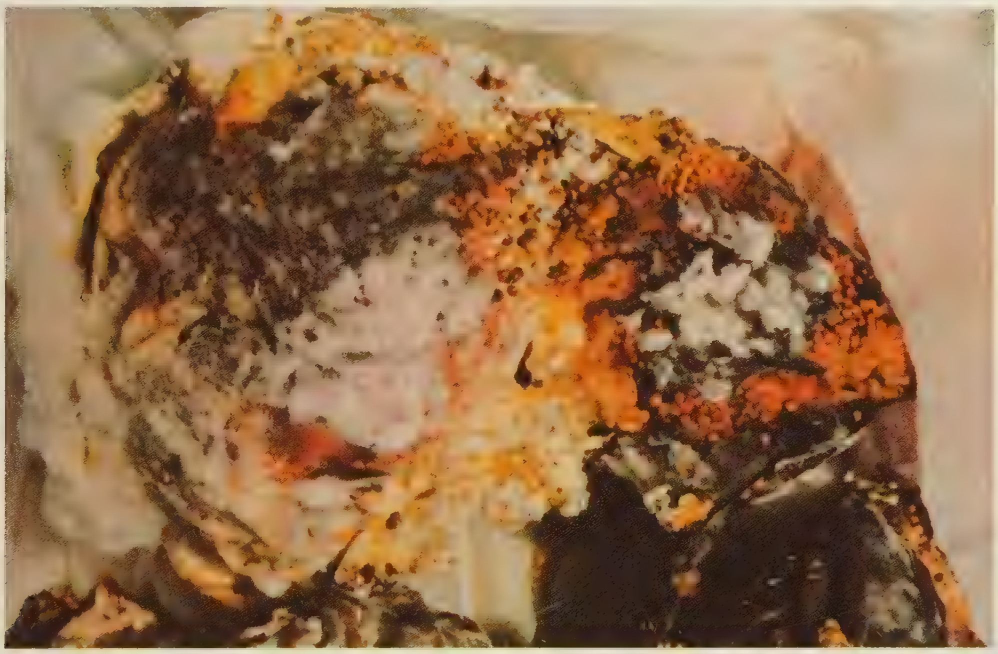
347 Scab



349 Scab

SCLEROTIUM ROT is caused by *Corticium rolfsii* (page 255). The fungus is common in warm temperate and subtropical regions, and thrives in well-aerated moist soils. It can cause 'damping off' of seedlings if conditions permit, or alternatively it may attack mature roots. Slightly infected roots may not be noticed during harvesting and grading and, if refrigeration is not applied, a rapid decay ensues, with copious formation of spherical resting bodies (sclerotia) (page 287). The author has observed severe sclerotium rot in beets imported into the UK from Cyprus, following a failure to cool them on board ship. This fungus is a notifiable disease organism in the UK, and infected consignments are subject to statutory action. Control has been achieved by harvesting the crop early before the weather becomes hot enough to favour development of the fungus (6).

WATERY SOFT ROT may be caused by *Sclerotinia minor* or by *S. sclerotiorum* (page 47), both of which are active at moderate temperatures and under wet conditions, whether through rain or irrigation (7). Beet roots are attacked in the field in Israel (7) and decay continues after harvest. Control is by cultural methods designed to reduce the number of persistent resting bodies (sclerotia) in the soil. Beets harvested from infested soil should be carefully graded, rapidly cooled, and marketed without undue delay.



350 Sclerotium rot

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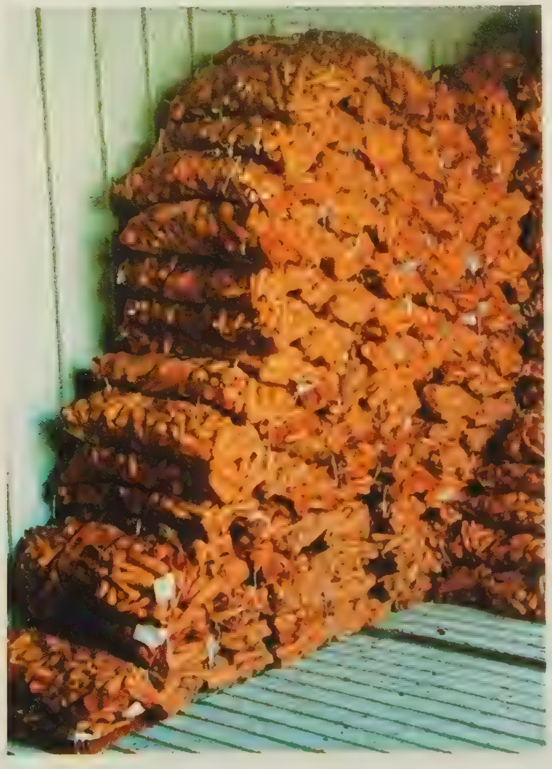
Carrots and parsnips

The **carrot**, *Daucus carota* L., originated in central Asia and spread in early times to the Mediterranean region. The wild form, ssp. *carota*, was used by the ancients as a medicinal plant; like other members of the family Umbelliferae (e.g. fennel, parsley), the carrot produces seeds rich in aromatic oils. The tap root of the primitive carrot was purple, until a yellow mutant arose (devoid of anthocyanin); both types spread east and west during the Middle Ages. At the beginning of the 17th century, in the Netherlands, repeated selection resulted in carrots with fleshy orange roots, and these carrots provided the basis for modern cultivars of ssp. *sativus*. The orange colour is due to ß-carotene, the precursor of vitamin A, making carrots a valuable dietary item; they also contain vitamins B and C and are high in sugars (9). Fresh carrot juice is popular in the Middle East, and raw carrot is a healthy salad dish. The more traditional use is as a cooked vegetable, an accompaniment to meat and an ingredient of soups and stews. Cultivars are classified according to whether the root is long or short, conical or cylindrical, tapered or stump-ended. Certain types are suitable for processing, by dehydration, freezing or canning.

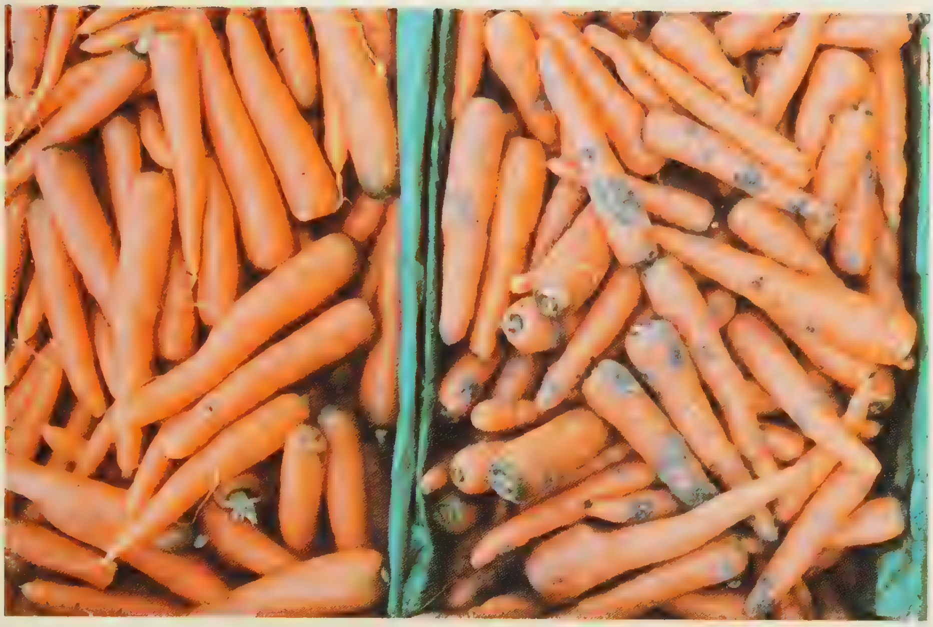


351 Pattern of sprouting in cargo of improperly chilled carrots (sluggish airflow, and hence substantial temperature gradient, from deck to deckhead)

Total world production is about 13 million tonnes, the top ten producers being China, the USSR, the USA, Poland, Japan, the UK, France, Italy, Germany and Canada. Carrots may also be grown in the tropics and subtropics at high altitude or during the cool season.

The plant is a biennial which, unless grown for seed, is harvested in its first year before the roots develop a woody core. Immature 'baby' carrots are especially prized, both for processing and for the fresh market. Young carrots are sometimes marketed in bunches, the foliage still attached. This looks very attractive initially, but the green tops transpire so rapidly that they soon wilt, having meanwhile taken up much moisture from the roots. If bunched carrots are to be held for more than a few hours they should be pre-cooled; this permits a 2-week storage period. The use of crushed ice is beneficial during transport and marketing. In order to minimise moisture loss and consequent flaccidity of the roots, young carrots are more usually topped as soon as the plants are harvested, whether by machine or by hand (10). (If untrimmed carrots are left on the field, for example during a lunch break, serious moisture loss can occur.) The topped roots are washed, graded, sorted and packed in nets, wooden trays, or directly into perforated polyethylene pre-packs. If carrots are thoroughly pre-cooled (e.g. by hydro-cooling) before being packed, the packaged roots may be held for several weeks at 0°C and high humidity.

Mature carrots are sometimes left in the ground over winter, the tops covered with straw as protection from frost. While cheaper than cold storage, this practice can lead to significant quality loss, especially if there is severe frost or if wet spring weather delays the harvest (3). Increasingly in temperate countries the carrot crop is harvested before the onset of winter, and held in refrigerated storage, sometimes for 6 to 8 months. Close trimming is essential if long-term storage is intended, since, if much stalk tissue is left, re-growth of shoots is facilitated. Leaf debris should, as far as possible, be excluded from storage, since it usually harbours disease organisms and can also be a source of ethylene. The topped roots are usually washed, and may also be drenched in a fungicide before being stored in bulk or in wooden crates. If licorice rot (q.v.) is likely to be a problem, it may be advisable to allow a brief curing period at ambient temperature and high humidity, to promote wound healing. Optimal storage conditions are a temperature of 0°C and a saturated atmosphere, it having been established that a relative humidity of 98 to 100% gives significantly better results than one of 95% (2). By preventing moisture loss from the roots, high humidity storage not only preserves quality but also reduces susceptibility to invasion by fungi. Ideal

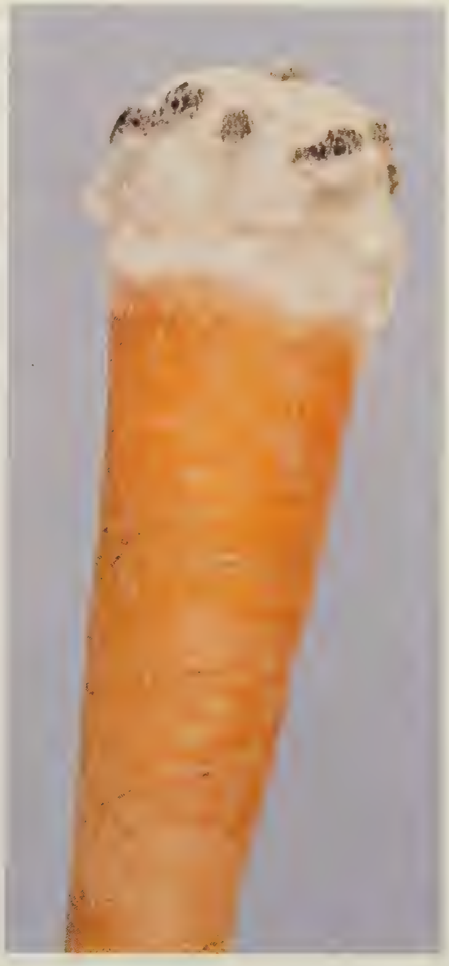


352 Chalaropsis and thielaviopsis rots (right) of carrots

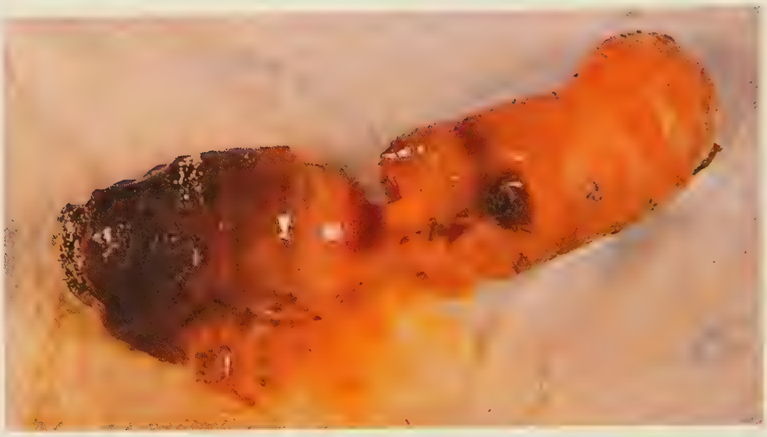
storage conditions can be provided by a moist-air cooling system such as the ice-bank, which has an air-water counterflow system capable of delivering saturated air at a temperature very close to 0°C. As well as minimising weight loss and decay, this storage environment effectively prevents re-growth (the development of shoots and roots) and is generally superior to conventional cold storage (3). Low temperature also delays the onset of bitterness, a physiological disorder related to senescence. Bitterness is induced by ethylene, hence the importance of segregating carrots from sources of ethylene such as apples, pears, or any decaying vegetation (8).

Even under optimal conditions there is, after a few months, a visible deterioration in 'skin-finish' known as 'silvering' (3). This reduces market value, especially of roots intended for supermarket prepacks. Provided that only slight deterioration has occurred, appearance can be restored (and is subsequently maintained) if the carrots are given a brief re-wash prior to packing (3).

Although in northern Europe refinements in storage have extended the availability of the home-grown crop, there is still a ready market in the spring for new carrots imported from warmer lands. In the same way that Bermuda can supply eastern Canada, so Cyprus and Israel supply the UK and Scandinavia. In the Mediterranean trade the freshly harvested roots are washed, sorted and packed in cotton nets or, if pre-cooling is available, they may be packed in perforated polyethylene sacks. Shipment is generally in conventional refrigerated vessels, although container carriage is sometimes used. The required carriage temperature is usually given as 1°C, as a



353 Watery soft rot of carrot



354 Bacterial soft rot of carrot

precaution against inadvertent freezing (which would occur at about −1°C). If carrots arrive for loading at ambient temperature it is essential to cool them as rapidly as possible; the aim should be to reduce the air delivery temperature below 5°C within 24 hours of loading, and to 1°C within 3 days of loading, without ever allowing it to fall below 0°C. Not all refrigerated ships are suited to this task; the ideal design includes small, independently refrigerated holds with under-deck air delivery. For optimal cooling efficiency the fresh air vents should be kept closed for the first three days, with minimal intake subsequently. In refrigerated containers it may be more practical to stipulate that fresh air vents be set at the start of the voyage to give minimal intake throughout. An appropriate setting would be such as to provide the equivalent of no more than one quarter of an air change per hour (relative to the volume of the chamber when empty). Excessive introduction of fresh air is a common error. Not only is such a practice unnecessary for carrots (and many other cargoes), it has distinct disadvantages; the introduced air is often warm and moist, thereby increasing the heat load and the rate at which moisture condenses (and freezes) on the cooling coils. A more moderate ventilation policy is required for efficient refrigerated carriage.

The **parsnip**, *Pastinaca sativa* L., is in the same family as the carrot, but is less widely grown. It is essentially a temperate crop, and is especially appreciated in the UK, Germany, the Netherlands, Canada and the northern USA. The pale, sweetly aromatic roots can be stored for up to 6 months, optimal conditions being a temperature of 0°C coupled with a saturated atmosphere (2). The high soluble sugar content (which tends to increase further during storage) causes a substantial depression in freezing point, so that parsnips do not freeze until about −2°C. Except for canker (q.v.), parsnip diseases (4) are similar to those of carrots.

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BACTERIAL SOFT ROT of carrots and parsnips caused by
***Erwinia* spp.**
***Pseudomonas* spp.**

OCCURRENCE Bacterial soft rot can occur on carrots and parsnips from any location, for example the USA (1,3) the USSR (6), New Zealand (2), Italy (8) and Romania (9). The causal organisms include *Erwinia carotovora* ssp. *carotovora* (Jones) Dye (1,2, 4,7,8,9,10), *Erwinia chrysanthemi* pv. *chrysanthemi* Burkh., McFadden & Dimock (10), *Pseudomonas marginalis* pv. *marginalis* (Brown) Stevens and *Pseudomonas marginalis* pv. *pastinacae* (Burkh.) Young, Dye & Wilkie (3). *Erwinia* spp. are not important in cold-stored roots but can cause heavy losses at ordinary temperatures (7). *P. marginalis*, on the other hand, can develop even under refrigeration.

SYMPTOMS Lesions may occur anywhere on the root, though sometimes infection occurs predominantly at the crown or the root-tip whence it progresses rapidly through the core region (3,10). There is usually a distinct line of demarcation between healthy and rotted tissue. Infected flesh is often brownish in colour, and may be extremely soft and slimy or (in parsnip) firm and hard (3,4). The presence of secondary bacteria frequently results in the production of a putrid odour. Secondary infections by fungi often mask the primary cause.

BIOLOGY Bacteria survive on plant debris in the soil (6), and may infect the growing crop (10), especially after heavy rains or in badly drained fields. Rotting can occur in field-stored roots (8) or during transport and storage. Roots are likely to be surface-contaminated at the time of harvest, and the likelihood of post-harvest infection and decay is largely determined by post-harvest handling practices and the storage environment. *P. marginalis* can cause rotting even at 0°C, while *Erwinia* spp. are generally insignificant below about 5°C (the optimal growth temperature for these bacteria being approximately 30°C). A build-up of bacteria in washing tanks can lead to severe rotting of roots, especially those packed in polyethylene and held at ambient temperature (7).

CONTROL If field infection is a problem it may be necessary to improve drainage. Contamination can be reduced by washing harvested roots in chlorinated water, which should be renewed frequently and checked regularly for its disinfectant capacity (7). If the roots are subsequently to be kept at ambient temperature it is advisable to allow thorough surface drying, the bacteria being very sensitive to desiccation (2); similarly, bright sunlight is lethal to these organisms (4). In recent times, however, it has become more usual to use refrigeration, and if roots are kept at temperatures close to 0°C then erwinia rot may be controlled without recourse to chlorination or surface-drying (5). Even a period of a few days at low temperature can be beneficial, in that it appears to confer resistance in roots subsequently removed to higher temperatures (7). On the other hand, decay caused by *P. marginalis* is inhibited, but not halted, by low temperature storage, and infected produce cannot be held for long.

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BLACK ROT of carrots and parsnips caused by

Alternaria dauci **(Kühn) Groves & Skolko**
Alternaria radicina **Meier, Drechsler & Eddy**
(syn. *Stemphylium radicinum* (Meier, Drechsler & Eddy) Neergaard)

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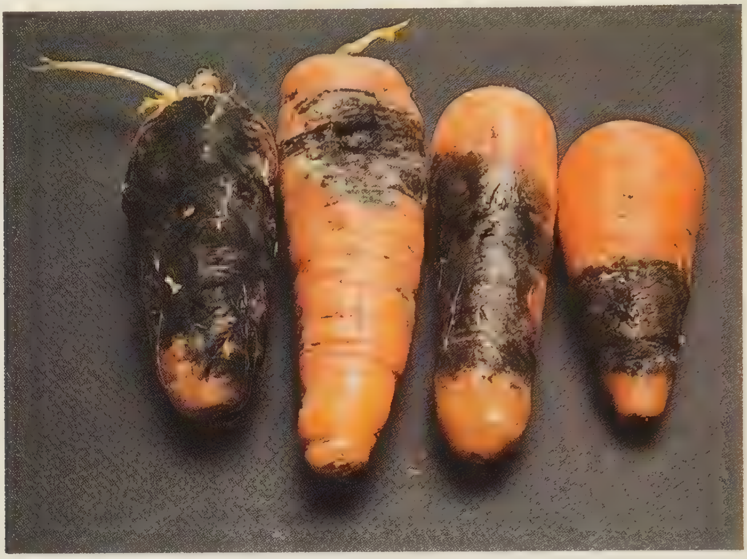

OCCURRENCE The most usual cause of black rot is *A. radicina*, recorded in many countries, including Canada (1), the USA (5,7), Switzerland (2), Poland (3), Finland (10), the UK (6) and New Zealand (9). Accounts of the disease differ (1,4,5,6,9). *A. dauci* is usually restricted to the leaves, but there are reports from Norway and Germany (8) that roots may be attacked during storage.

SYMPTOMS There may be small black superficial lesions ('root-spotting'), often located at root-scars, or a more extensive black rot on the crown or side of the root (4). A definite black margin separates diseased and healthy tissue, and lesions enlarge rather slowly even under optimal conditions. In a humid atmosphere characteristic dark spores may form on the surface, and microscopic examination serves to distinguish black rot from canker (q.v.) and licorice rot (q.v.).

BIOLOGY Species of *Alternaria* are seed-borne (6,10) and have thereby been distributed to new areas (9). Plants which survive the seedling stage may exhibit infected leaf stalk bases and crowns. Once the soil is infested, the fungus proliferates on crop debris, and spores (conidia) are disseminated by air currents. *A. radicina* can cause a serious foliage blight similar to that incited by *A. dauci* (9). More usually, however, *A. radicina* attacks the leaf stalk bases and thence the crown. In addition there may be direct infection (from the soil) of any part of the root, especially where there are cracks or splits (6). Infection of uninjured roots can also occur but it is a slow process and the resulting lesion is very shallow (5). Harvested carrots and parsnips may show no symptoms and yet undergo rotting in store, even at temperatures close to 0°C (5). However, the decay does not usually spread from root to root in store. In the few instances when *A. dauci* has been found in root lesions, the source has been traced to diseased foliage debris. Besides being unsightly, black rot lesions predispose stored roots to secondary infection by the grey mould fungus (q.v.).

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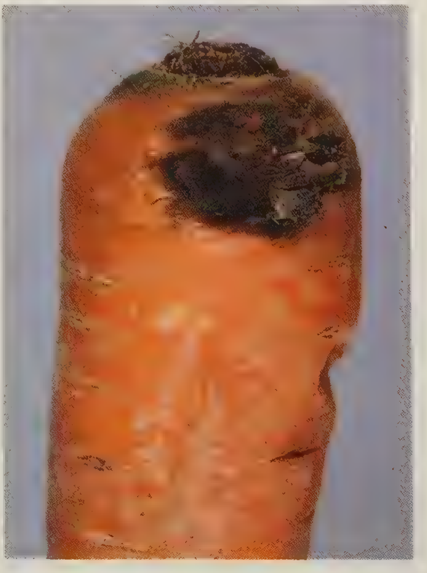

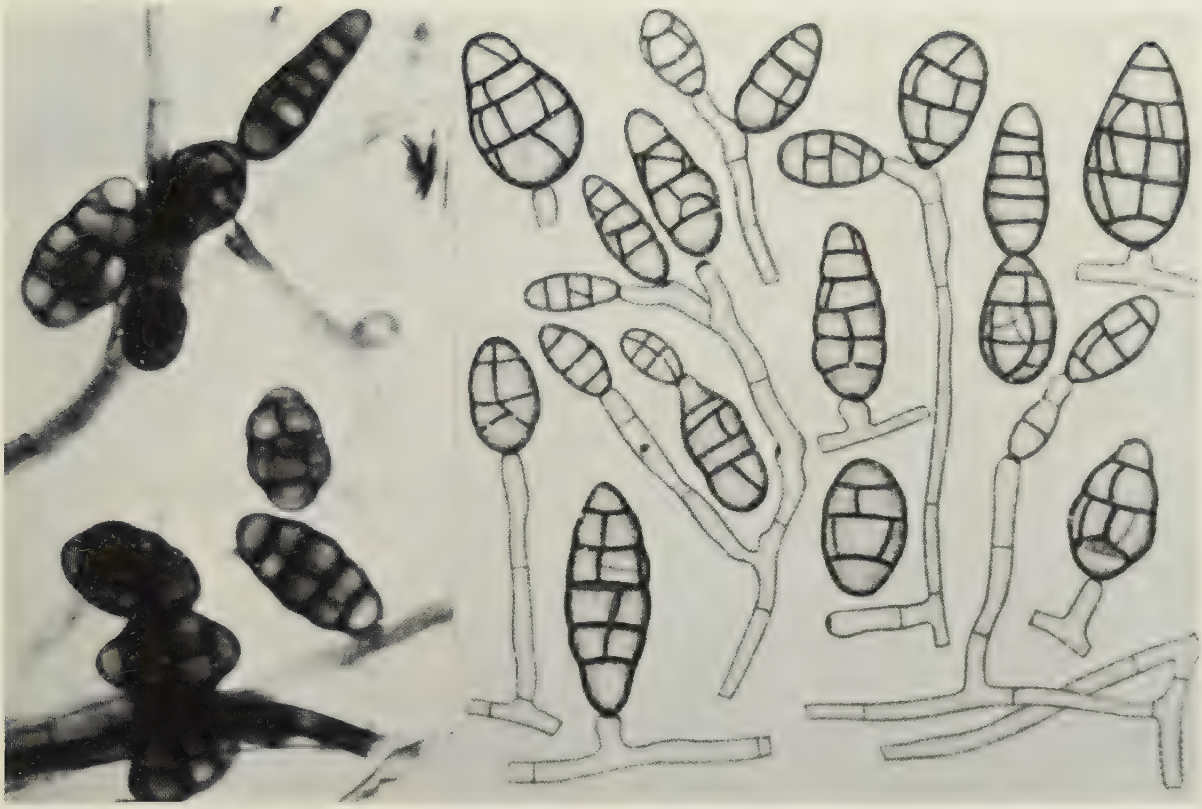

357 *Alternaria radicina* (from CMI Descr. No. 346, Ellis & Holliday 1972) Conidia and conidiophores × 500

CONTROL It is important to ensure that the seed is free of disease, and this can be achieved by soaking it for several hours in a heated fungicide bath (6). Crop debris should not be left on the soil surface, but removed or ploughed in as soon as possible after harvest (9). Fungicide treatment reduces post-harvest rotting (3). Crop rotation can be helpful, but it is necessary to exclude other umbelliferous crops such as parsley and celery, since they are also susceptible to infection by this fungus (2,9).

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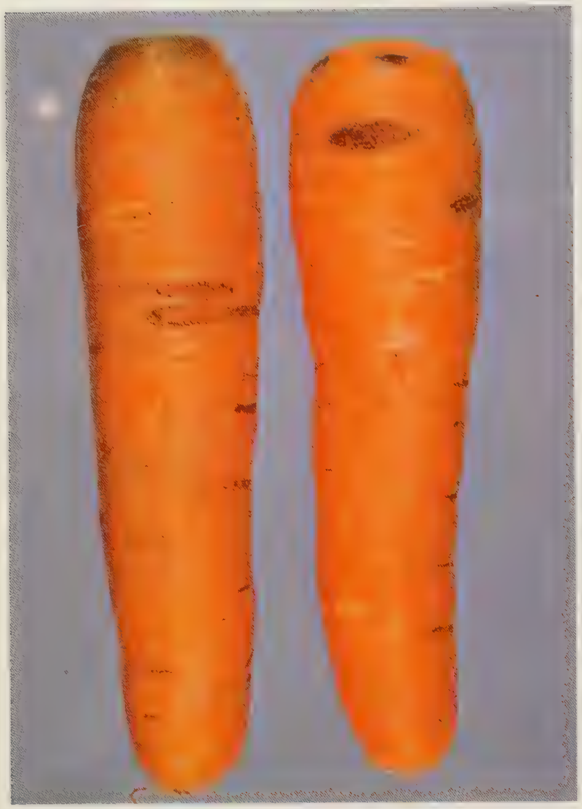
CAVITY SPOT of carrots and parsnips – a disease complex

358



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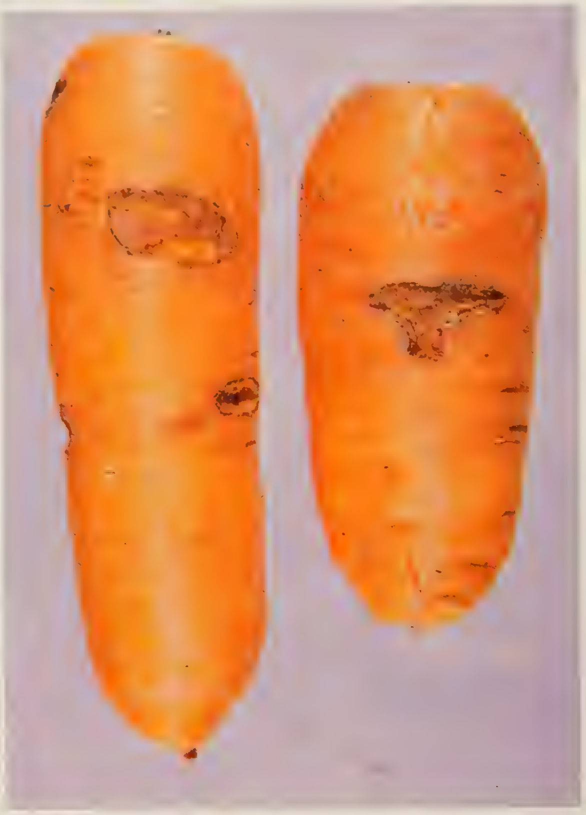
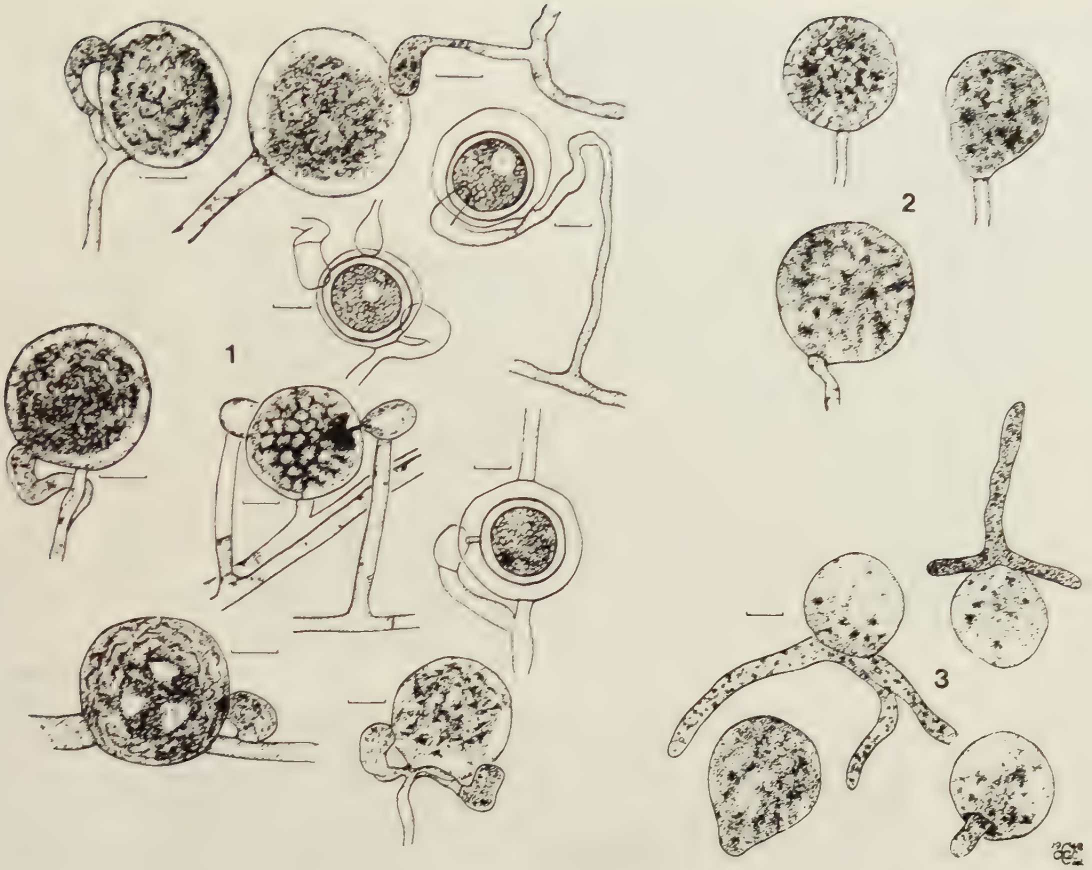


OCCURRENCE Cavity spot constitutes a significant blemish and can result in serious economic loss, both in the fresh market and processing industries (2,3, 5). It is common in carrots but rare in parsnips (4). Investigations have been carried out in the UK (1,2,3,10), Norway (7), Israel (5,9) and the USA (4,8).

SYMPTOMS Lesions may be small (1–2 mm) and almost circular, to substantial (up to 1 cm) and elongated transversely to form lens-shaped cavities (2). The 'skin' over the larger lesions tends to rupture, leaving a ragged fringe (4), and the interior of the cavity is greyish in colour. When roots have been peeled for processing, even the small lesions may still be apparent, as dark blemishes with raised borders (2).

BIOLOGY Cavity spot develops in the growing crop and is evident at harvest time. The causes are complex, and several hypotheses have been advanced, including calcium deficiency (8), other aspects of mineral nutrition (5), insect attack, and infection by bacteria (9). Incidence is often very irregular within a field (1), but in general tends to be highest during wet seasons and in poorly drained soils (4,9). Recent work has shown that cavity spot, together with associated root die-back, can be controlled with certain fungicides (1,7), and that symptoms can be induced by species of *Pythium*, notably *P. sulcatum* Pratt & Mitchell and *P. violae* Chesters & Hickman (3,6,10). It seems probable that a range of organisms may be capable of causing similar symptoms, and that the disease syndrome develops only under certain environmental conditions (2,9).

CONTROL Effective control measures were devised even before the cause had been elucidated. Incidence of cavity spot can be substantially reduced by growing the crop on narrow ridges rather than on wide flat beds (5); the significant factor is probably the increase in aeration, which would favour the plants more than the disease organisms. Control has also been achieved through soil sterilisation, and the use of a fungicide as seed-treatment, soil drench and foliar spray (1,2,7,10). Cavity spot tends to be less prevalent in alkaline soils; at pH 8 and above, fungicide treatment may be unnecessary or a reduced rate of application may be sufficient (10). Further recommendations include crop rotation and the breeding of resistant cultivars (9).



360 *Pythium violae* (from *Trans. Br. mycol. Soc.* **27**, 55–62, Chesters & Hickman 1944) 1 oogonia, antheridia, and resultant thick-walled oospores 2 sporangia 3 germinating sporangia (Bar = 10 μ)

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CHALAROPSIS and THIELAVIOPSIS ROTS of carrots and parsnips caused by

***Chalaropsis thielavioides* Peyr.**
(syn. *Chalara thielavioides* (Peyr.) Nag Raj & Kendrick)
***Thielaviopsis basicola* (Berk. & Br.) Ferraris**
(syn. *Chalara elegans* Nag Raj & Kendrick)

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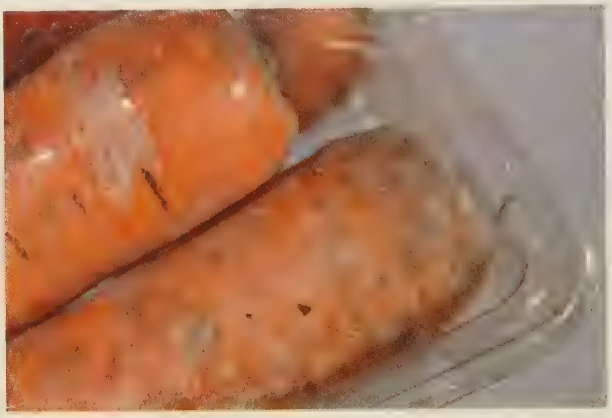

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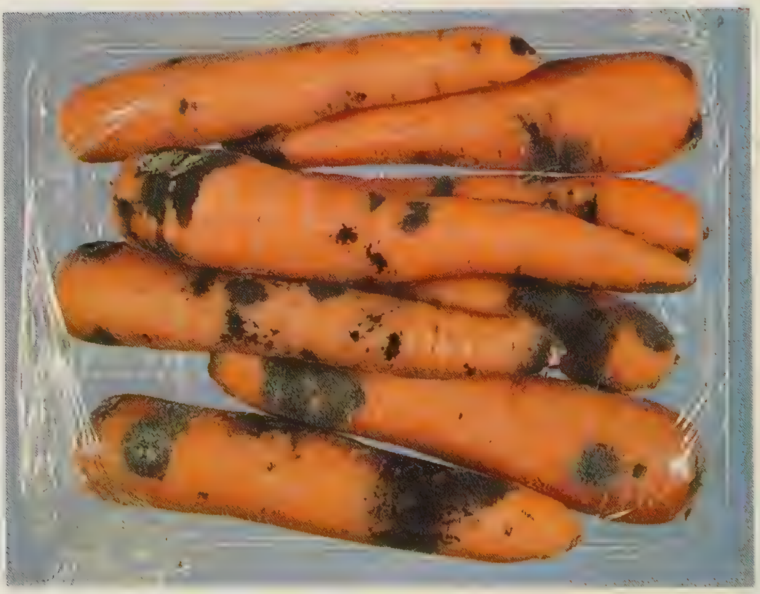
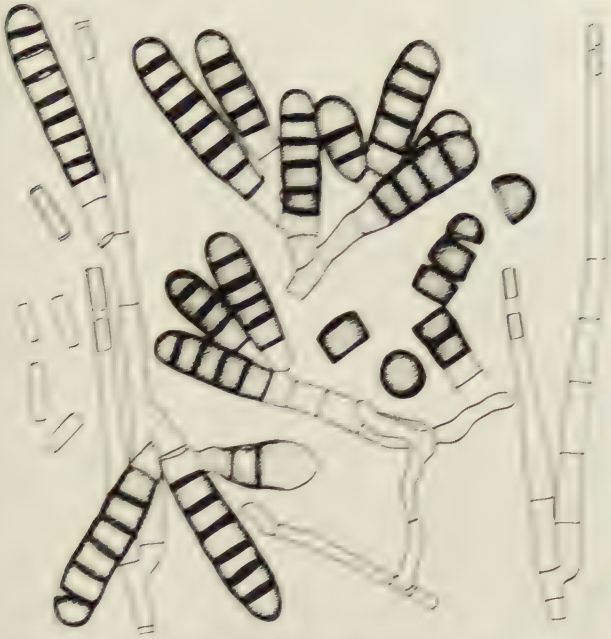


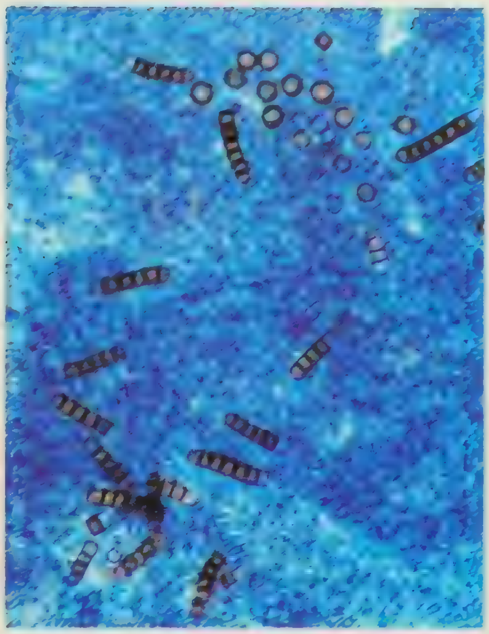
OCCURRENCE Chalaropsis rot of carrots has been recorded in the Netherlands (2), Italy, Sweden, Norway (1), Denmark (5), the UK (3), Germany (7,8), Czechoslovakia (10) and the USA (6). Thielaviopsis rot has been found in the USA (4), Italy and Norway (1), and the author has observed it in French and English carrots.

SYMPTOMS The two fungi frequently exist together on carrots and initially produce similar symptoms. The first sign is a very fine growth of mould, which later becomes dark grey and powdery. In mature lesions *C. thielavioides* may be distinguished by its deep black colour. Rotting is superficial, but the dark patches constitute a serious blemish which can render a consignment worthless (3,4).

BIOLOGY Both moulds survive in plant debris or as resting spores in the soil (8,9). Carrots grown in infested soil are contaminated at the time of harvest and, if storage conditions are warm and humid, the fungi proliferate rapidly, especially at wound sites (1,8). There is copious spore-production, firstly pale thin-walled spores (endoconidia) and later dark thick-walled spores (macroconidia) which become resting spores (chlamydospores). Severe losses



363 *Thielaviopsis basicola* (from CMI Descr. No. 170, Subramanian 1968) Endoconidia, endoconidiophores and chlamydospores × 500)



364 Chlamydospores of *Thielaviopsis basicola* (stained with cotton blue in lactophenol)

have occurred in mature carrots stored in clamps (7,8) and in young carrots intended for pre-packs (3). These fungi have become important since the introduction of polymeric films (e.g. polyethylene and PVC) as liners or covers for bulk bins and as bags and overwraps for pre-packaged carrots (2). Such materials are ideal if used in conjunction with refrigeration, but at higher temperatures there is increased likelihood of mould growth (3).

CONTROL Once a field or packing station is contaminated it is difficult to control these organisms because of their tolerance of fungicides (3,5). The main precautionary measures are careful harvesting and handling of carrots to minimise surface abrasion (8), and the use of refrigeration, especially if film wrapping is to be used (1,5). Temperatures below 5°C are necessary to prevent the growth of these moulds.

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CRATER ROT of carrots caused by
Athelia arachnoidea **(Berk.) Julich**
Sclerotial state: *Rhizoctonia carotae* Rader

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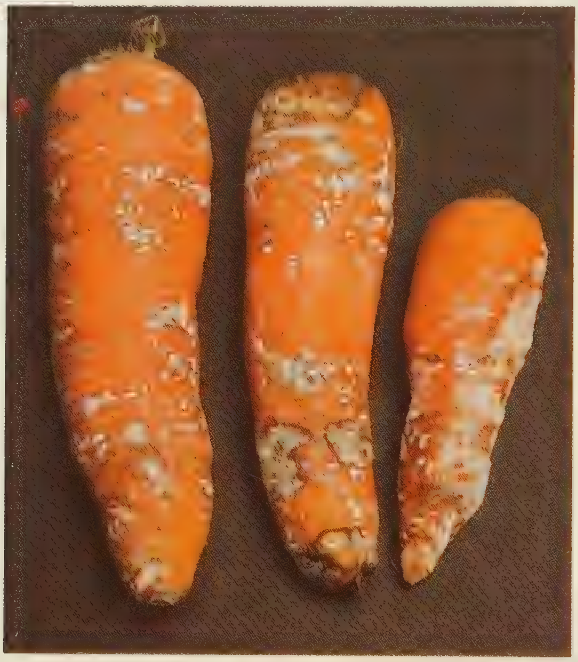

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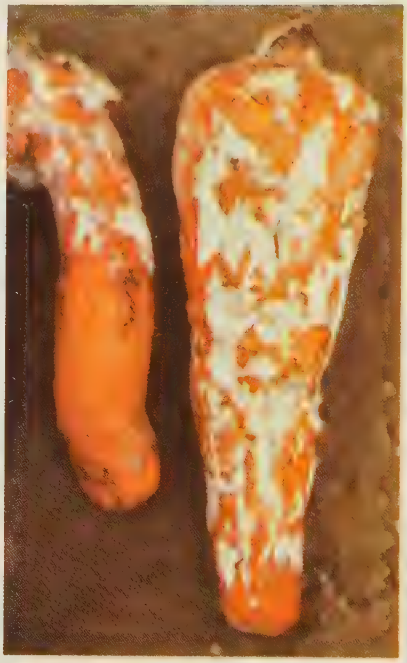

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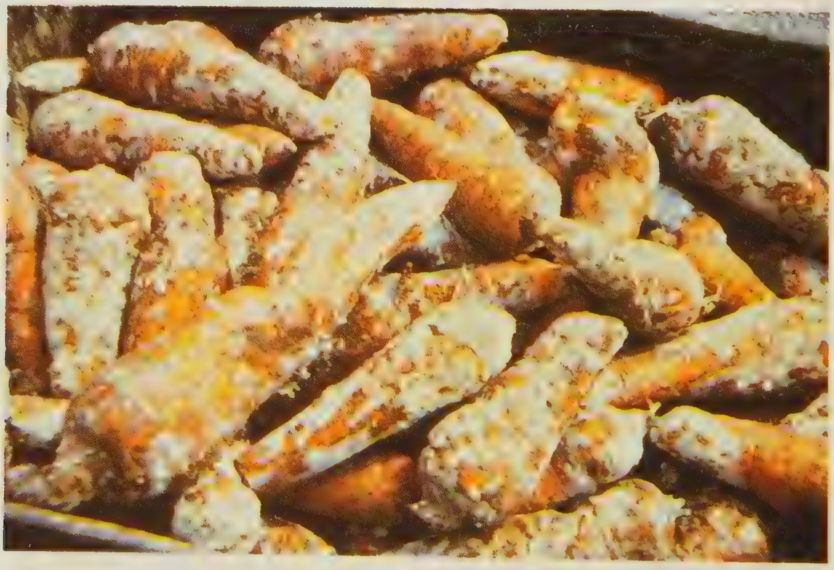
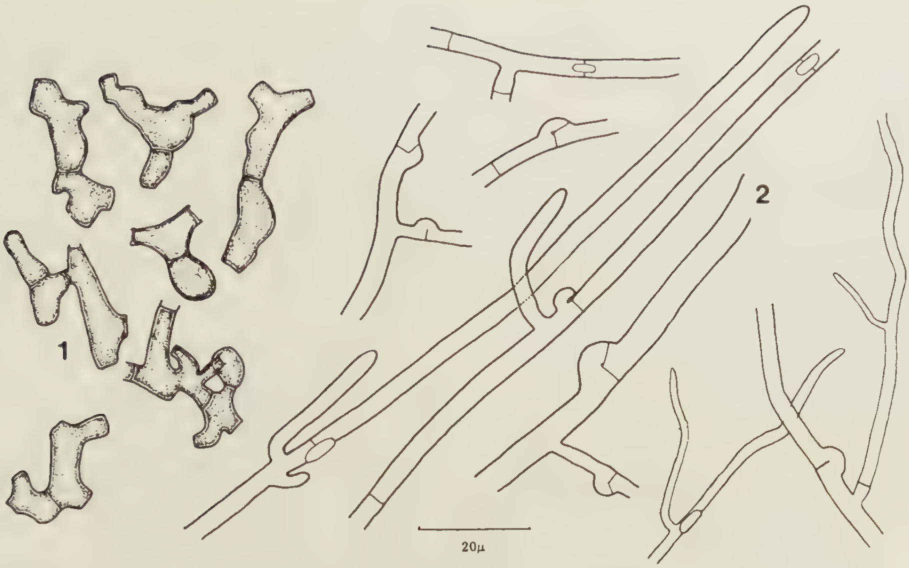


OCCURRENCE Crater rot has caused serious losses in the USA (7,8,10), the UK (3), Norway (2), Sweden (9), Denmark (5) and the USSR (4). It seems to occur only on carrots held under refrigeration and at high humidity, and has not been found on carrots in common storage (8). In the processing industry economic loss results from the need for extra hand-paring of carrots disfigured by deep lesions (10).

SYMPTOMS Small whitish tufts of mould appear on the roots after several weeks of cold storage (8). Pits develop beneath the lesions, enlarging into sunken 'craters', and fluffy white mould spreads over the roots (9,10). In old craters the mould may have a crystalline appearance, and small (1 to 3 mm) dark brown to black resting bodies (sclerotia) are occasionally found (2,7).



368 *Rhizoctonia carotae* (from CMI Descr. No. 408, Mordue 1974) 1 sclerotial cells 2 hyphae, showing clamp connections

BIOLOGY The fungus originates in the soil, and the sexual state (a hymenium giving rise to basidiospores) has been found on decaying leaves (1). It is the sclerotial state, however, which is usually encountered on carrots. Harvested roots may be carrying incipient infection (7); another important source is contaminated wooden storage boxes (6). Symptoms of decay in the carrots generally become evident only after two or three months (7). Mould development is favoured by high humidity, and in a moist atmosphere there is substantial growth even at temperatures close to 0°C (1,3,7,10). The presence of a film of water on the roots is especially conducive to fungal growth (8). Secondary invasion by grey mould (q.v.) and other fungi frequently occurs (8).

CONTROL Post-harvest fungicide dips may be ineffective against this fungus (3), and control depends on general sanitation and storage techniques. The aim should be to maintain a constant cold storage regime, avoiding temperature fluctuations which might lead to condensation (7). If the source of infection is storage boxes, these can be treated by steaming or fumigation, or by lining the interior with clean polyethylene (6). Polyethylene also serves to minimise dehydration of the carrots, and is beneficial provided that refrigeration is sufficiently prompt and the flow of cooling air is not impeded.

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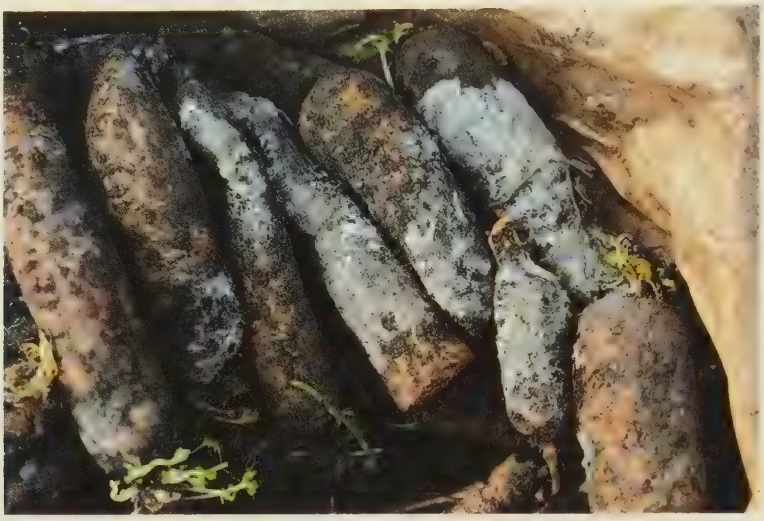
GREY MOULD ROT of carrots and parsnips caused by

Botryotinia fuckeliana **(de Bary) Whetzel**

Conidial state: *Botrytis cinerea* Pers.

369



OCCURRENCE Grey mould rot is a major storage disease of carrots grown in temperate regions, for example the USA (6), Canada (8), the USSR (3), the UK (4,5) and Scandinavian countries.

SYMPTOMS Two main types of lesion may be produced. In carrots which are resistant to infection there is a delimited lesion, dark brown to black in colour and showing no obvious mould growth (5). By contrast, in carrots which have become susceptible there is a progressive invasion of the root, and the colonised tissue becomes water-soaked, then leathery, and covered with mould. Under cool humid conditions the mould may remain white, and the eventual formation of resting bodies (sclerotia) causes a slight resemblance to watery soft rot (q.v.). Usually, however, there is copious formation of characteristic grey-brown spores (conidia).

BIOLOGY The fungus persists in the soil as sclerotia, and also survives in crop debris, on which it produces abundant spores. Vigorous young plants are not attacked, but during the latter part of the growing season wet weather or irrigation can result in infection of dying foliage. This can occur by means of air-borne spores or through contact with soil or crop debris. The fungus may spread into the base of the leaf stalks and even into the crown of the root, though this rarely occurs before harvest. In addition, spores are probably washed into the crown depression. Thus when the roots are harvested they may be carrying incipient infection of the crown or leaf stalk base, or at least be contaminated with spores, infected leaf debris and soil (2,5). Freshly harvested uninjured carrots are resistant to invasion by this fungus (7), but they become susceptible if wounded or allowed to wilt (3). Root tips are often found to be infected, through being particularly prone to injury (4) and also, or alternatively, because of the rapidity with which this part of the root loses moisture by evaporation (1). If carrots are stored at

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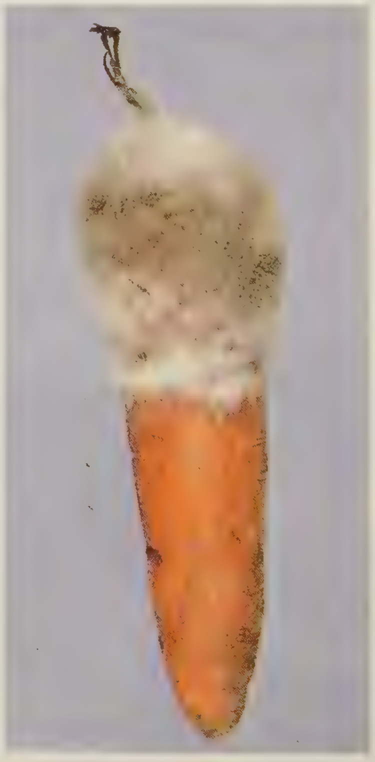

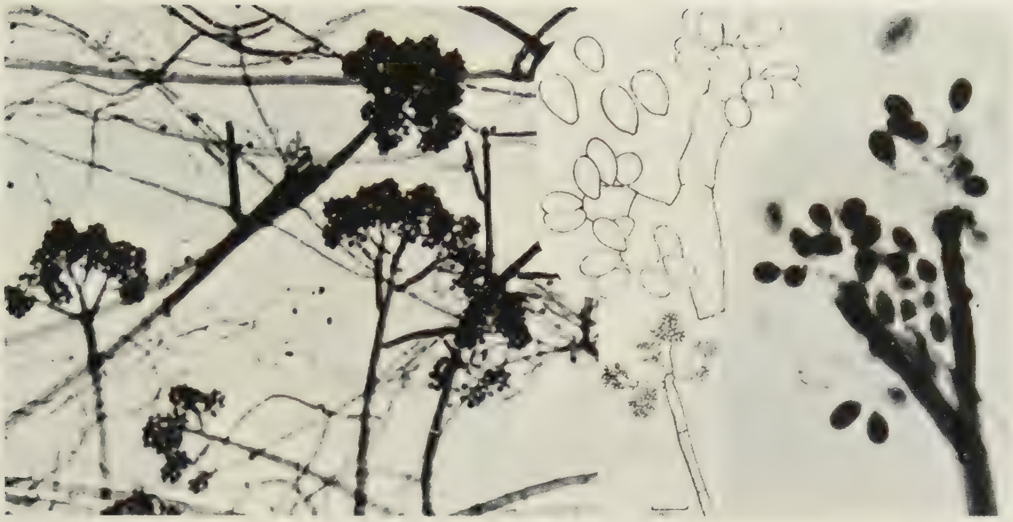

371 *Sclerotinia fuckeliana* (conidial state: *Botrytis cinerea*) (from CMI Descr. No. 431, Ellis & Waller 1974) Conidia and conidiophores × 500

temperatures above 5°C, and sprouting occurs, the new leaf growth is very susceptible to infection by grey mould, which then progresses into the crown (10). Even at 0°C *B. cinerea* is able to cause infection and rotting and, as the storage period is extended, an increasing percentage of carrots lose their resistance (5,7). The fungus is able to spread into adjacent sound roots by contact, and into more distant roots (if injured) by means of air-borne spores disseminated throughout the store (5).

CONTROL The crop may be sprayed with a systemic fungicide one day before harvesting (9). It is desirable to harvest in dry weather (especially if the roots are to be stored unwashed) in order to avoid bringing in excessive soil. Care should be taken to prevent unnecessary damage, and to minimise moisture loss (2). Post-harvest washing serves to eliminate soil and debris, and various fungicide treatments permit successful long-term cold storage (4,6,8), especially if a wet-air cooling system is used (page 268) (4).

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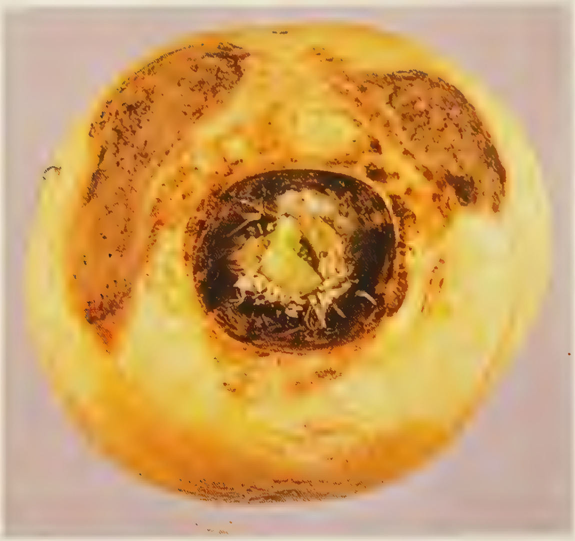
9 TAHVONEN R. (1985) The prevention of *Botrytis cinerea* and *Sclerotinia sclerotiorum* on carrots during storage by spraying the tops with fungicide before harvesting. *Annales Agriculturae Fenniae* **24,** 89–95.

10 VIRGIN W.J. (1942) Investigation of storage diseases of carrots. *Phytopathology* **32,** 830 [Abstract].

ITERSONILIA CANKER of parsnips caused by
Itersonilia pastinacae **Channon**

372



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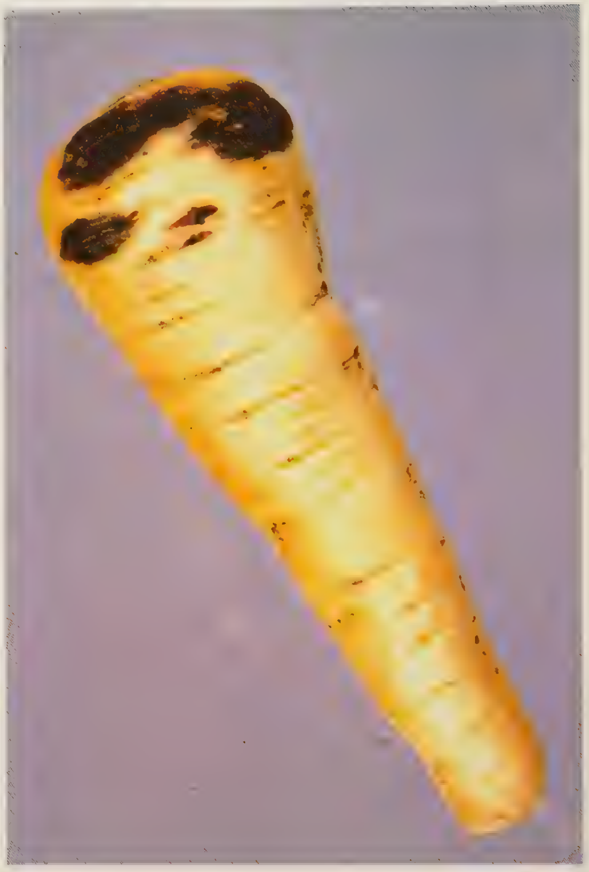
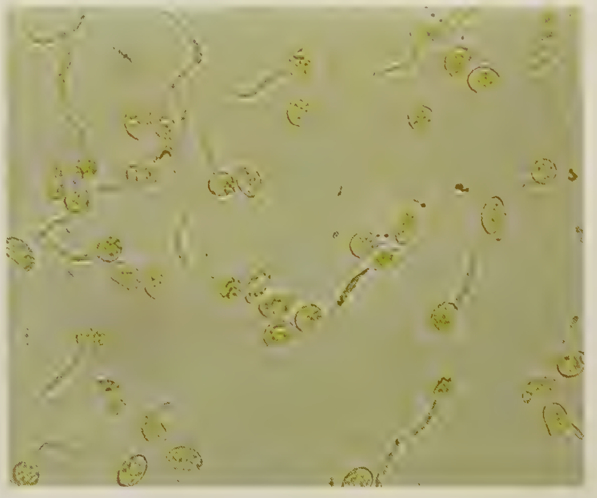


OCCURRENCE This is the most important disease of parsnips, and has been recorded in the UK (2), the USA (6,10), Canada and Australia (9). It does not appear to affect carrots, but has been found on dill and wild parsnips (CMI Map 523).

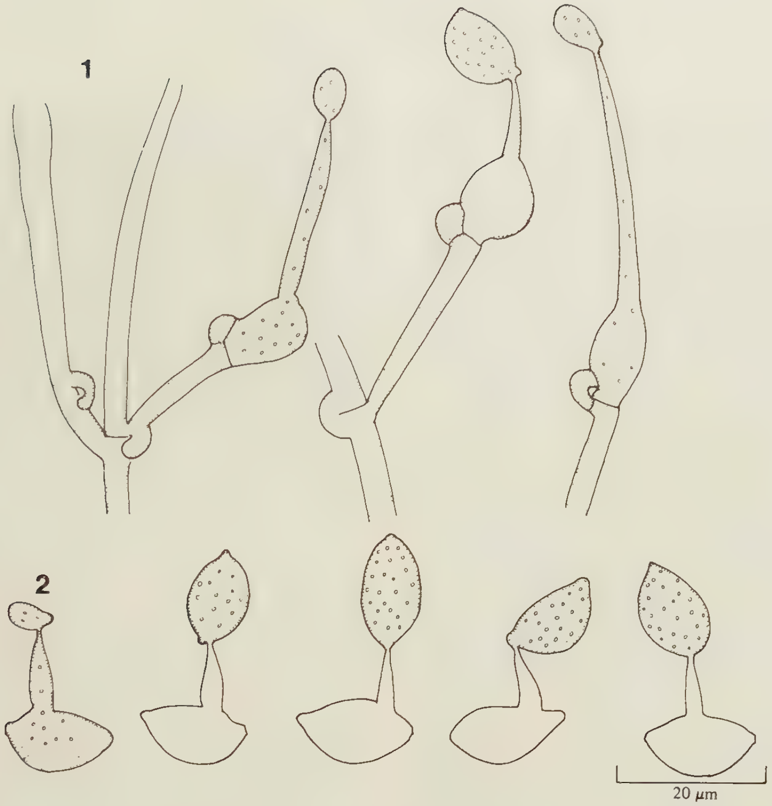
SYMPTOMS Cankers commonly occur on the crown or shoulder of the root, but may also be found on the shank, as lens-shaped lesions associated with lateral root bases. Affected tissue is dark brown or black, often with a purplish tinge (2). Lesions may remain fairly shallow, but are frequently invaded by secondary soft-rot bacteria which cause rapid breakdown of tissue (9). Black canker-like lesions are also symptomatic of black rot (q.v.), licorice rot (q.v.) and phoma rot (q.v.), and microscopic examination is therefore necessary (2,6,8,10).

BIOLOGY The fungus survives as resting spores (chlamydospores) in infected parsnip roots left in the ground (7). In cool moist conditions air-borne spores (ballistospores) (5,7) are discharged from exposed infected tissue and impinge on the foliage of the new parsnip crop, causing leaf-spots (3). It is probable that spores produced on leaf lesions are washed down by rain and lodge in the crown depression or on the shoulder of the root, thence giving rise to cankers (10). The incidence of canker appears to be directly related to rainfall during the growing season (1). The fungus also attacks parsnip flowers and can be transmitted on or in the seed (3).

CONTROL Parsnip seed may be soaked in a fungicide (3) or subjected to a steam/air treatment to inactivate the fungus. Cankered roots should be removed from the soil before the new crop is planted (7). Since incidence of canker is related to time of sowing and spacing of plants, these factors can be



374 Germinating ballistospores



375 *Itersonilia perplexans* (from *Trans. Br. mycol. Soc.* **82,** 13–29, Webster, Davey, Duller & Ingold 1984) 1 primary ballistospores developing from clamped mycelial segments 2 secondary ballistospores developing by repetitious germination

manipulated so as to achieve maximal marketable yields (1). A fungicide spray can be used to control the leaf spot phase of the disease. Much effort has gone into breeding a canker-resistant parsnip (4); if a susceptible cultivar is used, then crop rotation may be advisable.

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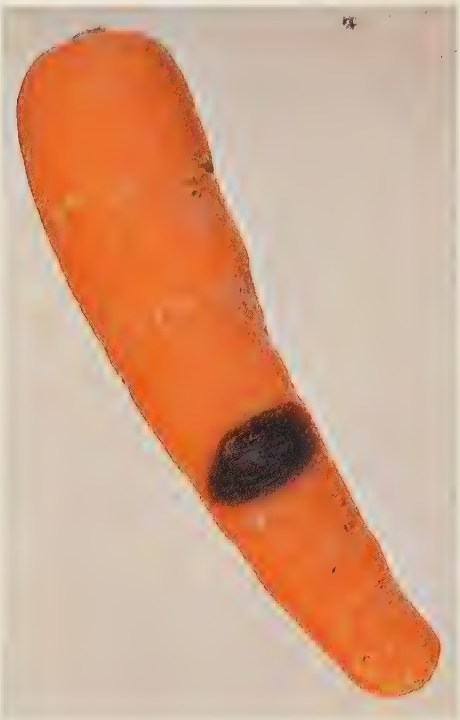
1 BROWN A., NOURISH W.H. & FLETCHER J.T. (1964) The effect of spacing and time of sowing on the incidence of parsnip canker. *Journal of Horticultural Science* **39,** 103–110.
2 CHANNON A.G. (1963) Studies on parsnip canker. 1. The causes of the diseases. *Annals of Applied Biology* **51,** 1–15 illus.
3 CHANNON A.G. (1969) Infection of the flowers and seed of parsnip by *Itersonilia pastinacae. Annals of Applied Biology* **64,** 281–288 illus.
4 CHANNON A.G., DOWKER B.D. & HOLLAND H. (1970) The breeding of Avonresister, a canker-resistant parsnip. *Journal of Horticultural Science* **45,** 249–256.
5 INGOLD C.T. (1984) Further observations on *Itersonilia. Transactions of the British Mycological Society* **83,** 166–174 illus.
6 OLIVE L.S. (1952) Studies on the morphology and cytology of *Itersonilia perplexans. Bulletin of the Torrey Botanical Club, New York* **79,** 126–138.
7 SMITH P.R. (1967) The survival in soil of *Itersonilia pastinacae* Channon, the cause of parsnip canker. *Australian Journal of Biological Science* **20,** 647–660 illus.
8 SOWELL G. & KORF R.P. (1960) An emendation of the genus *Itersonilia* based on studies of morphology and pathogenicity. *Mycologia* **52,** 934–945.
9 WALKER J. (1957) Diseases of parsnips. *Agricultural Gazette of New South Wales* **68,** 404–406 illus.
10 WILKINSON R.E. (1952) Parsnip canker is caused by *Itersonilia* sp. *Phytopathology* **42,** 23.

LICORICE ROT of carrots and parsnips caused by

Mycocentrospora acerina **(Hartig) Deighton**

376



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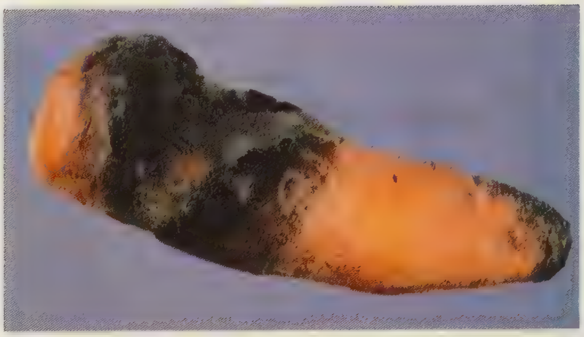
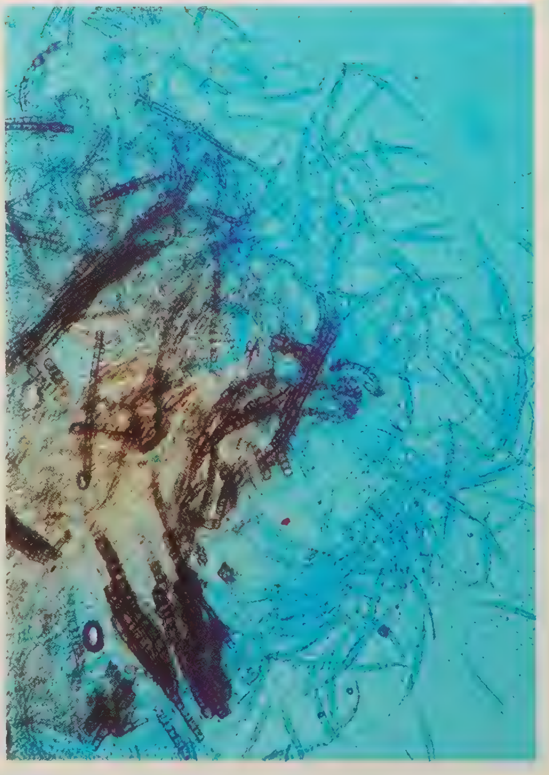


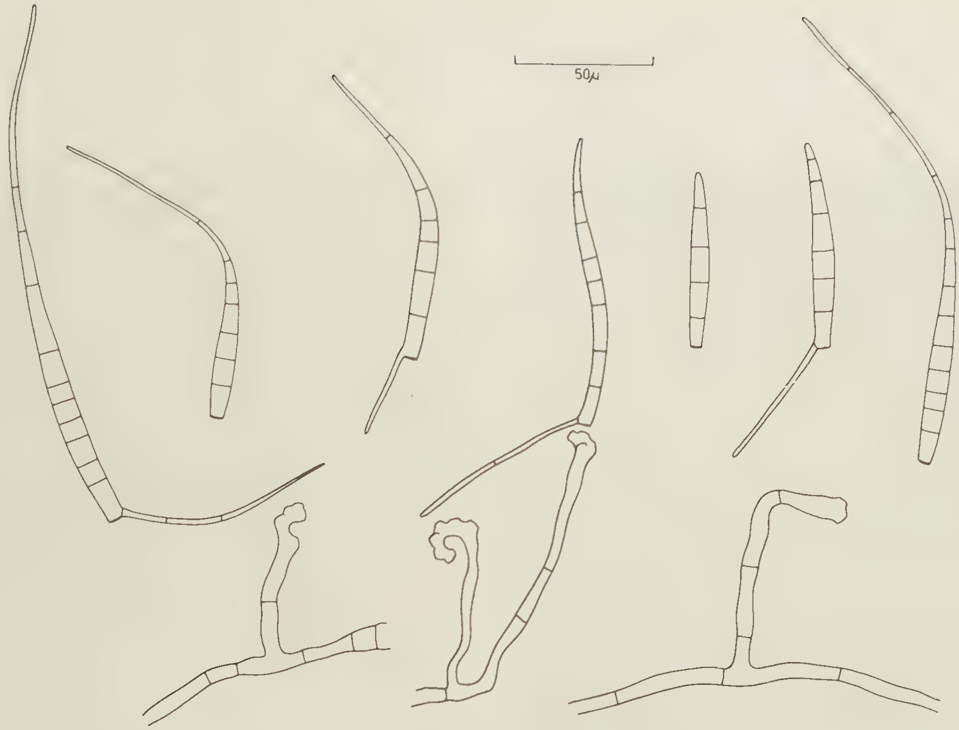
OCCURRENCE Licorice rot of carrots has long been known in northern Europe, and has caused losses in, for example, the UK (10), Norway (1) and Denmark (8). It also occurs in the US (8,9), Canada (3), Australasia and Japan. Records on parsnips include the UK (2) and Germany (6).

SYMPTOMS Lesions may occur on any part of the root; they are blackish externally and may be deeply sunken (9,10). In cross-section the rotted area is mainly black but has a diffuse water-soaked brown margin. Characteristic spores form on the surface of the lesion and, under humid conditions, there may be growth of dark mould tinged with orange. In the absence of this latter symptom, microscopic examination may be necessary to distinguish between licorice rot and black rot (q.v.) of carrots, and between the various canker diseases of parsnips (2).

BIOLOGY The fungus survives in the soil as dark thick-walled resting spores (chlamydospores), which germinate in the presence of carrot and parsnip crops. The fungus may also be present in streamwater used for irrigation (5). Mould strands colonise the surface of the developing tap-root, on which are formed more chlamydospores (4). Thin-walled spores (conidia) are less common, but may be found on leaf lesions after wet weather. If infection spreads to the base of the leaf stalk, then the crown of the carrot may eventually be invaded. However, infection is usually through wounds on the surface of contaminated roots. Mechanical harvesting has led to increased incidence of this disease (1). At the beginning of the storage period carrots show considerable resistance to fungal development, and lesions at first remain localised. After some months, however, the lesions become progressive and rotting may be severe even at 0°C (3), although there is no significant spread from root to root (4). Under warm conditions the fungus grows rapidly, the optimal growth temperature being between 17° (8) and 20°C (2).



378 Chlamydospores (brown) and conidia (stained blue)



379 *Mycocentrospora acerina* (from CMI Descr. No. 537, Sutton & Gibson 1977) Conidia and conidiophores

CONTROL Crop rotation can be helpful, but it is necessary to avoid planting other susceptible crops such as celery and parsley (2). Careful harvesting is of great importance, the aim being to minimise surface injury to the roots (1). Some damage is unavoidable, and a period of curing (page 268), even for half a day, allows significant wound-healing and consequent reduction in infection (7). Good control of licorice rot can be achieved by the use of a systemic fungicide as a post-harvest dip or drench.

1 APELAND J. (1974) Storage quality of carrots after different methods of harvesting. *Acta Horticulturae* No. 38, 353–357.

2 CHANNON A.G. (1965) Studies on parsnip canker. 4. *Centrospora acerina* (Hartig) Newhall – a further cause of black canker. *Annals of Applied Biology* **56,** 119–128 illus.

3 CHEESEMAN G.N. & ANDERSON M.G. (1985) *Mycocentrospora acerina* associated with storage rot of carrot in Atlantic Canada. *Plant Disease* **69,** 177 [Abstract].

4 DAVIES W.P., LEWIS B.G. & DAY J.R. (1981) Observations on infection of stored carrot roots by *Mycocentrospora acerina. Transactions of the British Mycological Society* **77,** 139–151 illus.

5 IQBAL S.H. & WEBSTER J. (1969) Pathogenicity of aquatic isolates of *Centrospora acerina* to carrots and parsnips. *Transactions of the British Mycological Society* **53,** 486–490.

6 KLEWITZ R. (1972) Schäden an Pastinak durch *Centrospora acerina* (Hartig) Newhall. *Nachrichtenblatt des Deutschen Pflanzenschutzdienstes* **24,** 166–168 illus.

7 LEWIS B.G., DAVIES W.P. & GARROD B. (1981) Wound-healing in carrot roots in relation to infection by *Mycocentrospora acerina. Annals of Applied Biology* **99,** 35–42.

8 NEERGAARD P. & NEWHALL A.G. (1951) Notes on the physiology and pathogenicity of *Centrospora acerina* (Hartig) Newhall. *Phytopathology* **41,** 1021–1033.

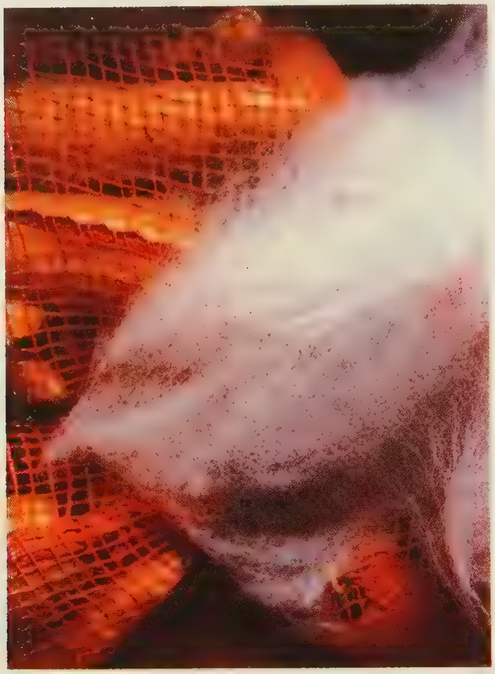
9 RADER W.E. (1945) *Ansatospora acerina* found causing decay of stored carrots in Wayne County, New York. *Plant Disease Reporter* **29,** 522.

10 SRIVASTAVA S.N.S. (1958) Studies on *Centrospora acerina* (Hartig) Newhall, the cause of licorice rot of carrot. *Transactions of the British Mycological Society* **41,** 223–226 illus.

RHIZOPUS ROT of carrots and parsnips caused by

Rhizopus oryzae Went & Prinsen-Geerligs
Rhizopus stolonifer (Ehrenb. ex Fr.) Lind

380



OCCURRENCE Rhizopus rot may be found if roots are stored or transported without adequate refrigeration; with the increased use of cold chain facilities this disease has declined in importance. It used to be prevalent on carrots in the USA (1); more recent observations include those made in India, Romania (4) and Cyprus (3).

SYMPTOMS Infected tissue is pale brown, water-soaked and soft, and there may be copious exudation of liquid. Individual white mould strands can be distinguished with the naked eye, especially those of *R. stolonifer* which are coarse in comparison with those of *R. oryzae*. Both fungi give rise to globular spore-heads (sporangia) which are initially white and later black (1) (pages 81, 373).

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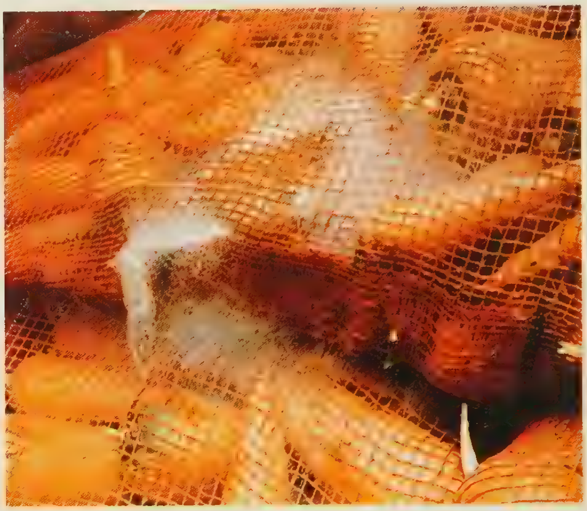

BIOLOGY *R. oryzae* is commonly found in soil, where it may persist as resting spores (chlamydospores). Different strains of *R. stolonifer* may combine and give rise to the sexual stage (zygospores). Both species produce abundant asexual spores (sporangiospores) which are blown about in the atmosphere (3) and can cause infection of moribund material (5). Harvested carrots are likely to be contaminated at the time of harvest but show a marked resistance to infection (2). If, however, roots are allowed to lose 8% by weight of their moisture then *R. stolonifer* (and probably *R. oryzae*) is able to invade (5). The optimal temperature for decay is approximately 25°C for *R. stolonifer* and 35°C for *R. oryzae*; neither fungus grows well below 10°C.

CONTROL The chief means of control is to cool the carrots as rapidly as possible after harvest (4). If refrigeration is not available then efforts should be directed towards the prevention of wilting, both during harvest and in store.

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1 BROWN J.G. (1950) Diseases of carrots. *Bulletin of Arizona Agricultural Experiment Station* No. 227, 19 pp illus.

2 MENKE G.H., PATEL P.N. & WALKER J.C. (1966) Physiology of *Rhizopus stolonifer* infection in carrot. *Zeitschrift für Pflanzenkrankheiten, Pflanzenpathologie und Pflanzenschutz* **71**, 128–140.

3 STOBO K.M. & KERKIDES C.V. (1974) Sources of infection in Cyprus carrots. *Phytopathologia Mediterranea* **13**, 133–138.

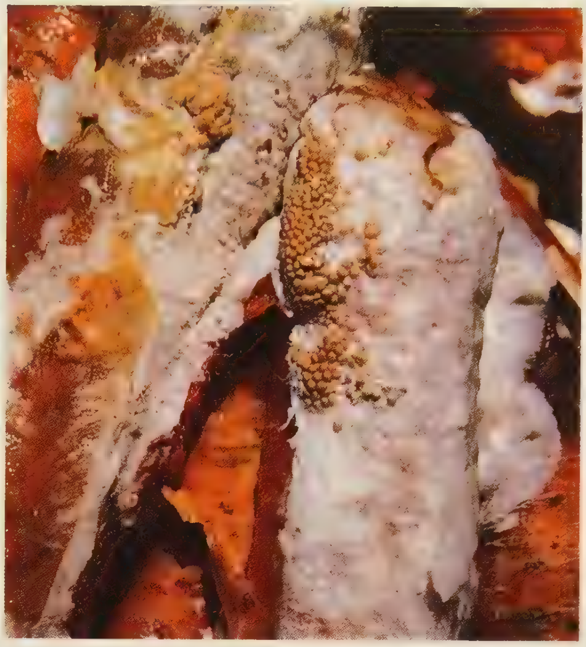
4 TAŞCĂ G., CERNĂIANU A., HULEA A. & WEKERLE J. (1975) [Methods for maintenance of quality of carrots during storage.] *Producţia Vegetală, Horticultura* **24**(8), 40–42.

5 THORNE S.N. (1972) Studies of the behaviour of stored carrots with respect to their invasion by *Rhizopus stolonifer* Lind. *Journal of Food Technology* **7**, 139–151.

SCLEROTIUM ROT of carrots and parsnips caused by ***Corticium rolfsii*** **Curzi**

Sclerotial state: *Sclerotium rolfsii* Sacc.

382



OCCURRENCE This disease is generally confined to warm temperate and subtropical regions, and has been recorded on carrots in the southern USA (3, 4, 5), Venezuela, Dominica, Bermuda, India, Israel, and parts of new Zealand and Australia (2). In Tasmania the fungus occasionally attacks carrots and parsnips but only during very warm weather (1).

SYMPTOMS A bright white silky mould spreads over infected roots, and has a characteristic fan-shaped appearance. Numerous spherical bodies (sclerotia) are produced, 1 to 2 mm in diameter and white at first, later shrinking slightly and turning brown. Infected tissue is soft and moist (3, 4, 5).

BIOLOGY This fungus is capable of attacking a very wide range of crops, and persists in the soil on organic debris or as sclerotia (2, 3). The sexual stage (a hymenium giving rise to basidiospores) is rarely formed, and is probably not important in infection (4). Under warm wet conditions mould strands from sclerotia grow over the soil and invade leaf stalk bases and subsequently the crown of the root (4). Roots may rot completely in the field (3, 5). If infection occurs shortly before harvest, however, the disease may not be manifest until the carrots are marketed. Growth of the fungus is insignificant below 10°C, its optimal growth temperature being approximately 30°C. Under favourable conditions mould growth is extremely rapid, and adjacent roots are soon invaded.

CONTROL This disease is controlled mainly by cultural methods. Since, in the presence of a susceptible crop, the fungus grows particularly vigorously at or just below the soil surface, it is advisable to practise deep-ploughing before the new crop is sown, the aim being to ensure burial of infected organic debris and sclerotia (3). Susceptible weed hosts should also be destroyed. The use of soil fumigants to kill sclerotia is not usually justified economically; solarization (page 132) is a cheaper and effective alternative. In the absence of specific control measures between crops, it has been noted that a period of prolonged dry weather, followed by rainfall or irrigation, can lead to decay of the sclerotia before the new crop is planted. Where sclerotia persist for many years, the land may be deemed useless for vegetable-growing, the best solution being to plant one of the few resistant crops such as strawberries (2). In less severely infested fields, carrots may be rendered more resistant to infection by application of a calcium fertilizer within 50 days of planting (3). Certain fungicides are also effective in reducing disease incidence (3).

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1 ANON. (1961) Diseases of carrots and parsnips. *Tasmanian Journal of Agriculture* **32,** 63–67 illus.

2 CHAMBERS S.C., ROOZEN A. & BARTHA L. (1977) Reactions of strawberries and carrots to *Sclerotium rolfsii. Australasian Plant Pathology Society Newsletter* **6,** 4–5.

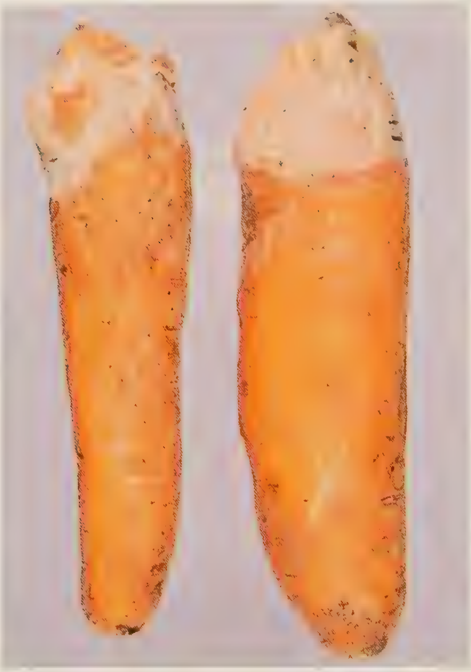
3 PUNJA Z.K., CARTER J.D., CAMPBELL G.M. & ROSSELL E.L. (1986) Effects of calcium and nitrogen fertilizers, fungicides, and tillage practices on incidence of *Sclerotium rolfsii* on processing carrots. *Plant Disease* **70,** 819–824 illus.

4 SMITH V.L., CAMPBELL C.L., JENKINS S.F. & BENSON D.M. (1988) Effects of host density and number of disease foci on epidemics of southern blight of processing carrot. *Phytopathology* **78,** 595–600.

5 WEBER G.F. (1931) Blight of carrots caused by *Sclerotium rolfsii,* with geographic distribution and host range of the fungus. *Phytopathology* **21,** 1129–1140 illus.

SOUR ROT of carrots caused by
Geotrichum candidum Link

383



384 Conidia of *Geotrichum candidum* (stained with cotton blue in lactophenol)

OCCURRENCE Sour rot may occur if carrots are held in moist conditions at high temperatures. The disease has been recorded in Mexican and American carrots (5), and has also been observed in Romania (4), Egypt (1) and India (3).

SYMPTOMS Rotting usually begins at the root tips, and decayed tissue is soft, watery and not discoloured (1). The surface of lesions becomes covered with off-white spores (conidia) and there is a distinctly sour odour of vinegar (5).

BIOLOGY The fungus is a common soil inhabitant, and occasionally causes serious rotting in the field (3). Even in a healthy crop, roots are likely to be contaminated at harvest. Wounding predisposes roots to infection (2), and the development of sour rot is particularly likely in a warm, moist, poorly ventilated environment. Such conditions occur in carrots which have been inadequately refrigerated , especially if they are packed in polyethylene (5). Decay is extremely rapid at temperatures above 20°C, whereas the fungus grows scarcely at all below 5°C.

CONTROL With regard to the disease in the field, some carrot cultivars are partially resistant (3). It may be advisable to improve drainage so as to prevent waterlogging. Post-harvest rotting is controlled by minimising injury to the roots, and ensuring that they are promptly pre-cooled and held under refrigeration. Fungicides are generally ineffective against *G. candidum.*

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1 EL-TOBSHY Z.M., ABDEL-SATTAR M.A. & BARAKA M.A. (1979) Sour rot, a post-harvest disease of certain vegetables in the A.R.E. *Agricultural Research Review, Cairo* **57**, 39–46.

2 LEWIS M.H. & SINCLAIR J.B. (1966) *Geotrichum candidum*: plant tissue pH and susceptibility to plant and animal isolates. *Plant Disease Reporter* **50,** 681–685.

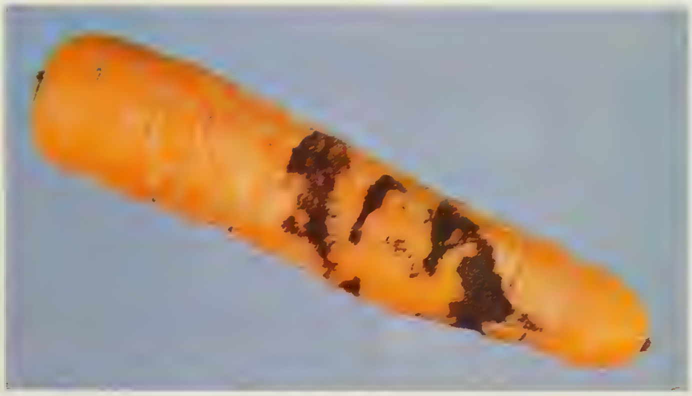
3 SUHAG L.L. & DUHAN J.C. (1979) *Geotrichum* rot of carrot – a new disease from India. *Indian Phytopathology* **32,** 642.

4 TAŞCĂ G. & HULEA A. (1972) [Mycoflora developing on carrots during storage.] *Analele Institutului de Cercetări pentru Protecţia Plantelor* **10,** 175–190.

5 WRIGHT W.R., SMITH M.A. & BERAHA L. (1964) Sour rot of carrots. *Plant Disease Reporter* **48,** 837–838.

VIOLET ROOT ROT of carrots and parsnips caused by *Helicobasidium purpureum* Pat.

Sclerotial state: *Rhizoctonia crocorum* DC. ex Fr.



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OCCURRENCE The disease is widespread in Europe and North America, and has received particular attention in the UK (1,2,4), Norway, Finland, France (3) and Canada (5). It is essentially a field disease but may also be encountered after harvest; Mukula (page 270) noted that, in one season at least, violet root rot was very common in consignments of Israeli carrots imported into Scandinavia.

SYMPTOMS Lesions are at first purplish in colour, turning cinnamon-brown with age. Infected tissue is firm, and later becomes covered with a leathery mass of small coalescing resting bodies (sclerotia) (5).

BIOLOGY Sclerotia can persist in the soil for several years (2), or the disease may be perpetuated in weed hosts or in susceptible crops such as parsley, celery, asparagus, sugar beet and potatoes (q.v.) (1). The fungus occasionally produces the sexual state (a hymenium giving rise to basidiospores), but it is mould strands growing directly from germinating sclerotia which are mainly responsible for infection (5). The optimal growth temperature lies between 20° and 25°C, and the fungus grows hardly at all below 5°C (5). Symptoms are not usually apparent in the crop until late in the growing season when, if infected roots are pulled out of the ground, an abnormally large mass of soil clings to the surface of the lesion. If an infected crop is left in the ground after reaching maturity there is a possibility of total loss, whereas if the roots are harvested immediately they can be stored with only a slight further development of decay.

CONTROL Since most commercial carrot cultivars appear to be susceptible to violet root rot (1) and few chemicals are effective against the disease (3), the main control measures involve crop husbandry. Early lifting may be advisable, before the disease becomes severe. Infected roots should not be ploughed back into the land, but removed and destroyed. Deep cultivation and generous applications of nitrogenous fertiliser help to reduce disease incidence, and it is important to eradicate susceptible weed hosts. Rotation with cereals can be worthwhile, as these crops are not attacked and the infectivity of the soil gradually decreases as the sclerotia germinate and then die (4). Work is in progress in the UK to identify sources of resistance in carrot cultivars.

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1 DALTON L.P., EPTON H.A.S. & BRADSHAW N.J. (1981) The susceptibility of modern carrot cultivars to violet root rot caused by *Helicobasidium purpureum*. *Journal of Horticultural Science* **56**, 95–96.

2 GARRETT S.D. (1949) A study of violet root rot. 2. Effect of substratum on survival of *Helicobasidium purpureum* colonies in the soil. *Transactions of the British Mycological Society* **32**, 217–223.

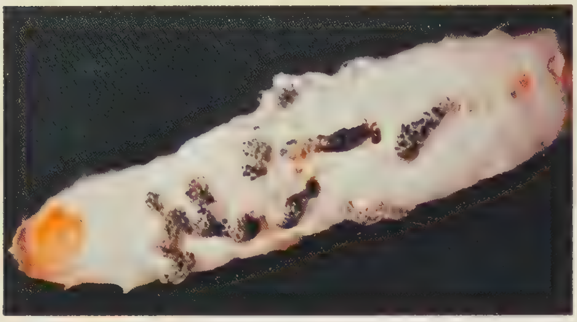
3 MOLOT P. & SIMONE J. (1968) Essais préliminaires de lutte chimique contre le Rhizoctone violet. *Phytiatrie et Phytopharmacie* **17**, 73–78.

4 VALDER P.G. (1958) The biology of *Helicobasidium purpureum* Pat. *Transactions of the British Mycological Society* **41**, 283–308.

5 WHITNEY N.J. (1954) Investigations of *Rhizoctonia crocorum* (Pers.) DC. in relation to the violet root rot of carrots. *Canadian Journal of Botany* **32**, 679–704 illus.

WATERY SOFT ROT of carrots and parsnips caused by
Sclerotinia minor **Jagger**
Sclerotinia sclerotiorum **(Lib.) de Bary**

386



OCCURRENCE *Sclerotinia sclerotiorum* is widespread in temperate growing areas and in subtropical regions possessing a cool wet season. Losses in stored carrots have been reported from Scandinavian countries, the USSR (5), Romania (9), the UK (4), Egypt (3), Israel, the USA, Canada (2) and Australia (1). Heavy losses used to occur in carrots shipped from Bermuda to Canada (10) and from Cyprus to the UK (7). Parsnips are also susceptible (4). A related fungus, *S. intermedia* Ramsey, now considered a form of *S. minor*, has been reported on carrots in the USA (6) and Japan (8).

SYMPTOMS Infected tissue is soft and watery but not discoloured. Mould appears on the surface, *S. sclerotiorum* being always pure white and *S. minor* being either white or buff-coloured. Primary lesions characteristically occur in the crown region, but during storage and transport the mould may spread by contact into all parts of neighbouring roots, causing 'nesting'. Irregularly shaped whitish bodies are formed and, after exuding drops of liquid, they darken and dry out, becoming black resting bodies (sclerotia). The sclerotia of *S. sclerotiorum* are generally within the range 2 to 20 mm, while those of *S. minor* from carrot are 1 to 3 mm.

BIOLOGY Sclerotia can persist in the soil for many years, surviving hot dry periods by remaining in a dormant state. A few weeks after the start of cool wet weather or irrigation the sclerotia 'germinate' in one of two ways. Those of *S. minor* usually give rise to a mass of mould strands. In Japan this fungus was observed on carrot plants under snow, causing crown rot despite temperatures around 0°C (8). The sclerotia of *S. sclerotiorum* typically produce the sexual stage (apothecia giving rise to ascospores). Spores are ejected into the air and impinge on the foliage. Infection by *S. sclerotiorum* becomes increasingly likely as the plants approach maturity; dense foliage tends to harbour drops of water, and under moist conditions dying outer leaves are susceptible to invasion. The fungus then grows down the leaf stalks to the crown of the root (4). Direct infection of the base of the leaf stalk may occur if ascospores are washed down into the crown depression (7). During wet seasons or in fields with excessive foliage the disease may be evident in the growing crop (10). Frequently, however, no symptoms are apparent at the time of harvest, and roots with incipient crown infection are sent for storage or export. In contrast to many other fungi, these species of *Sclerotinia* do not produce infective airborne asexual spores, and the only means by which the disease can spread in store is by direct contact of mould strands with adjacent roots. Hence the number of 'nests' in a consignment is a direct reflection of the level of infection at the time of harvest. The size of the 'nests' is determined by the storage environment. The strains of *S. minor* associated with carrots appear to be particularly adapted to low temperatures (6). Some strains of *S. sclerotiorum* can make significant growth at 0°C and are manifest if storage is prolonged for several months; at 20°C infected carrots and parsnips rot completely within a few days. With regard to the effect of relative humidity on sclerotinia rot, opinions differ as to whether roots should be kept dry (1,5) or moist (2). The author found that rotting of carrots was especially likely if they were allowed to wilt before being stored in a humid atmosphere (7).

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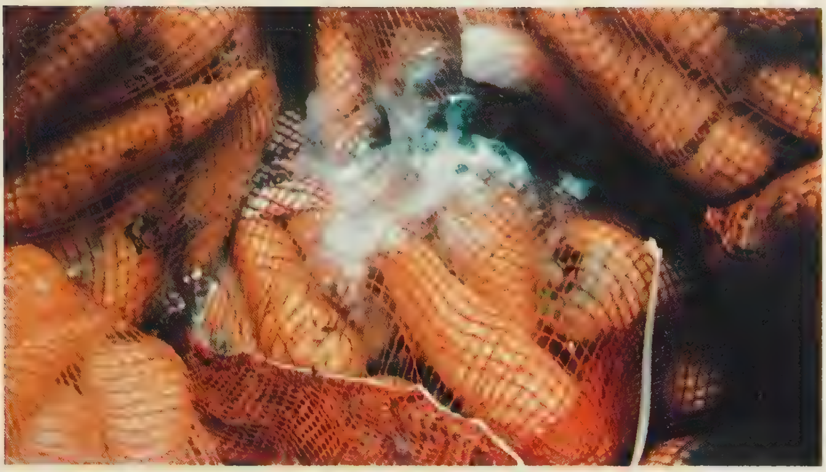

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CONTROL Careful fertiliser regimes can help to reduce the likelihood of infection; excessive nitrogen should be avoided, while potash is beneficial in that it increases the thickness of the natural corky layer on the root surface. Fungicide sprays are effective, but not always economically feasible. The control of weeds is important for several reasons. Firstly, many weed species can act as alternative hosts of *Sclerotinia* spp. (4). Secondly, weeds contribute to the density of foliage, and the resulting moist microclimate predisposes the carrot or parsnip leaves to infection (10). If manual labour is employed it is important to ensure that heaps of pulled weeds are not deposited on the crop, lest infection of the crushed foliage occur (7). Diseased plants should be uprooted, carefully removed and destroyed before more sclerotia form. These resistant resting bodies are very difficult to kill; for example the addition of sulphuric acid merely causes those of *S. sclerotiorum* to produce even more apothecia (4). Opinion is divided as to whether the land should be deep-ploughed so that sclerotia are buried (only to be turned up the following year) or shallow-ploughed in the expectation that, in damp soil, most will eventually undergo natural decay. Livestock should not be fed on infected foliage debris, since a percentage of sclerotia may survive in the droppings and transmit the disease to new pastures. It is reported that skilled labour can be used to advantage to grade out roots showing traces of crown rot (10). Rotting of roots in store can be minimised by the use of a post-harvest fungicide dip (9) and by prompt reduction of temperature to 0° or 1°C (7). Cultivars differ in susceptibility to this disease.

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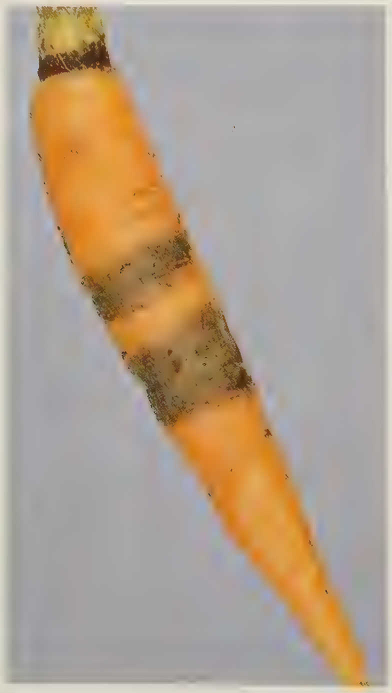
OTHER DISEASES of carrots and parsnips

ACROTHECIUM ROT has caused serious losses in Norwegian carrots (1). It is characterised by irregular dark brown to blackish lesions, the decay being firm and usually shallow. The optimal temperature for decay is approximately 18°C but the fungus, *Acrothecium carotae* Arsvoll, can also make significant progress at 0°C. It produces asexual spores (conidia) which can survive in the soil throughout the winter.

ASPERGILLUS BLACK MOULD ROT, caused by *Aspergillus niger* van Tieghem, is likely to be found only on roots kept at high temperatures (7). Wounds are necessary for infection, and black mould lesions are rapidly overgrown by other fungi.

BLUE MOULD ROT, caused by *Penicillium expansum* (page 88), has been reported on roots which have undergone excessive dehydration (6,7). The author has isolated *Penicillium chrysogenum* from the root tips of wilted carrots; lesions may also occur on the crown. The disease is usually of minor importance.

BROWN CROWN ROT and **RHIZOCTONIA CANKER** are caused by *Thanatephorus cucumeris* (page 307). Decayed tissue is fairly firm and dry, dark brown to black in colour, and clusters of irregular shaped resting bodies (sclerotia) 1 to 4 mm across may be produced on the lesion. Infection occurs in the field during warm weather (3,7), and multiple applications of fungicide are necessary for control. Some carrot cultivars are moderately resistant to the disease (3).



389 Fusarium rot

CANKER of parsnips (see also page 282) may be caused by species of *Phoma* (q.v.) or *Cylindrocarpon*. A serious black rot has been recorded on parsnips in the UK and in Ireland, the causal fungus being *Nectria radicicola* Gerlach & Nilsson (conidial state: *Cylindrocarpon destructans* (Zins.) Scholten).

CHARCOAL ROT, caused by *Macrophomina phaseolina*, is responsible for heavy losses of stored carrots in India. The fungus is common in tropical soils, and difficult to control (page 24).

DRY ROT of carrots may be caused by *Fusarium avenaceum* (Fr.) Sacc. , which can be transmitted in infected seed (7). Roots are attacked only if injured during harvesting and handling and, if no other organisms are involved, the lesions are characteristically limited in extent and leathery in texture, with no obvious mould. Frequently, however, *F. avenaceum* follows in the wake of other fungi such as *Alternaria radicina* (page 273), and in such mixed rots may give rise to white, yellowish or pinkish mould (6). Species recorded on parsnips include *F. sambucinum* and *F. sporotrichioides* (page 314).

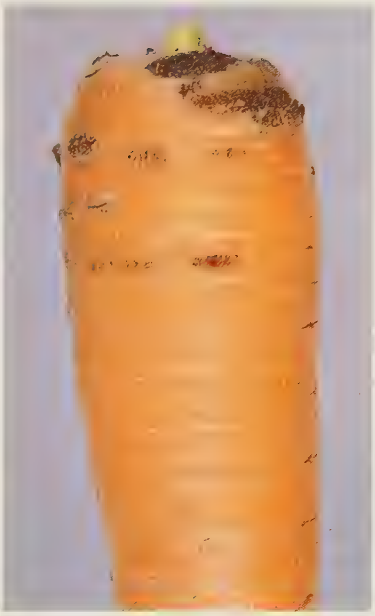
HARD ROT, caused by *Gliocladium aureum* Rader, may be indistinguishable from dry rot, the lesions being shallow, hard and leathery, and sometimes supporting brownish or pinkish mould growth (7). The disease has recently caused problems in cold-stored Brazilian carrots (8).

MUCOR ROT may be caused by *Mucor hiemalis* Wehmer. Affected tissue is soft and water-soaked, and generally supports a luxuriant growth of white mould rather similar in appearance to that of *Rhizopus* spp. (q.v.). *M. hiemalis*, however, can thrive at temperatures around 0°C, although it does not normally cause significant losses (7).

PHOMA ROT has been recorded on carrots in Scandinavia, *Phoma rostrupii* Sacc. occurring in Norway and Denmark, and *Phoma dauci* Mukula in Finland (6). Lesions are usually shallow, dark brown, and covered with greyish mould; occasionally minute black bodies (pycnidia giving rise to conidia) may be found. *P. rostrupii* has caused heavy losses in carrots grown for seed in the USSR. Species of *Phoma* can induce canker (q.v.) in parsnips. In Canada *Phoma complanata* (Tode ex Fr.) Desm. has caused severe losses, both in field and store, and it has been established that the fungus is seed-borne and can also persist in crop debris. Control measures include field hygiene, deep ploughing, crop rotation, and the use of a fungicide spray (2).

RUBBERY BROWN ROT may be caused by *Phytophthora cactorum* (Lebert & Cohn) Schroeter (7), *Phytophthora megasperma* Drechsler (10) or *Phytophthora porri* Foister (4). Infection occurs in the field, and losses in carrots have been reported from France, Canada (4), the USA and Australia (10). Firm, dark brown, water-soaked areas occur in wide bands on the root; affected tissue may remain rubbery in texture (7) or become soft (10). Rotting can progress in store even at 0°C and, under humid conditions, there is copious development of dense white mould. The disease is associated with water-logged fields, and the main control measure is therefore improvement of drainage.

RUBBERY SLATE ROT has been recorded on carrots in the USA (7) and may be caused by *Pythium debaryanum* Hesse or by *Pythium ultimum* Trow. Infection occurs during cool wet weather; affected tissue is soft and watery, turning from brown to greyish. Rotting can occur at low temperatures and, under humid conditions, there may be a white mould growth. Losses in store are not usually serious. Other species of *Pythium* cause cavity spot (q.v.).



390 Scab

SCAB is caused by a species of *Streptomyces* similar to the organism which causes common scab of potatoes (q.v.). Both carrots and parsnips may be attacked. Lesions are dark in colour and often raised above the root surface owing to abnormal proliferation of infected tissue. The disease has been recorded in, for example, the USA, the UK, Germany, Norway, the USSR and the Netherlands (5). Control measures include crop rotation, irrigation, and the maintenance of acid soil conditions. Scab symptoms have also been ascribed to the seed-borne bacterium *Xanthomonas campestris* pv. *carotae* (Kendrick) Dye, the cause of bacterial blight of carrots.

YEASTY ROT is caused by species of *Candida*, which thrive in warm moist conditions. The author found yeasty rot in carrots subjected to inadequate cooling and ventilation. Yeasts are often found in association with other fungi, for example *Sclerotinia sclerotiorum* (q.v.) (9).

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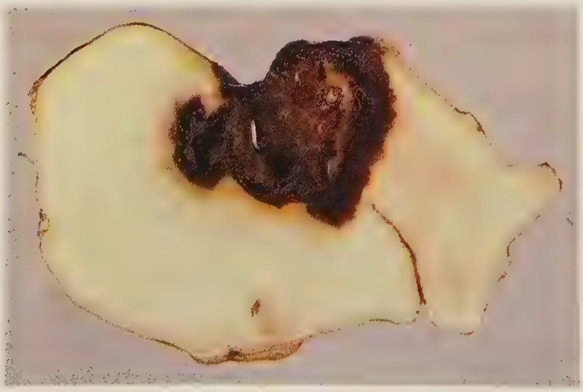
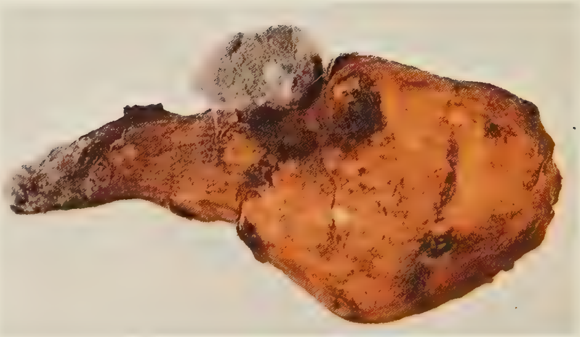
Jerusalem artichokes

The **Jerusalem artichoke**, *Helianthus tuberosus* L., is not really an artichoke, nor has it any connection with Jerusalem. Its taste is somewhat reminiscent of the globe artichoke (q.v.), but there the similarity ends. *H. tuberosus* is grown for its tubers, which can be used as food for man and animal. The plant is native to North America, and was introduced into Europe early in the 17th century (8). Its closest relative is the sunflower or girasole, *H. annuus* L. The accepted hypothesis, that 'Jerusalem' is here a corruption of 'girasole', does not stand up to scrutiny; it is suggested instead (Salaman, 1940) that the word which has been corrupted is Terneuzen, the Dutch port from which tubers were first despatched to England. An alternative name for the crop is sunroot; the produce industry meanwhile refers to sunchokes, a term which likewise has the virtue of brevity.

The crop has never achieved more than minor status, though there is currently an increased interest in using the tubers as a source of fructose sugar and alcohol (7,8). The plant thrives in a wide range of climates, from temperate to tropical. Research has been carried out in several countries, including the USA (4,8,10), Canada (7), New Zealand (1), the UK (5), France (3), the USSR (2,6) and Malaysia (9). The tubers are characteristically knobbled, and possess a delicate skin which is easily abraded. Unless great care is taken at harvest, there is a risk of physical damage leading to enhanced moisture loss (8,10). The resultant shrivelling is almost as great a hazard as post-harvest rotting (1,2,3,4,5,6,7,8,9). Deterioration can be minimised by careful handling followed by storage at 0°C and high humidity. Under such conditions tubers may be held for up to 4 or 5 months.

BACTERIAL SOFT ROT may be caused by *Erwinia carotovora* ssp. *carotovora* (page 271), which can gain entry through wounds (4). *Pseudomonas fluorescens* and *P. marginalis* characteristically act as secondary invaders following fungal infection (4,8). Decayed tissue is soft and slimy. Bacterial rotting is retarded, but not arrested, by storage temperatures close to 0°C.


391 Bacterial soft rot


392 Blue mould rot

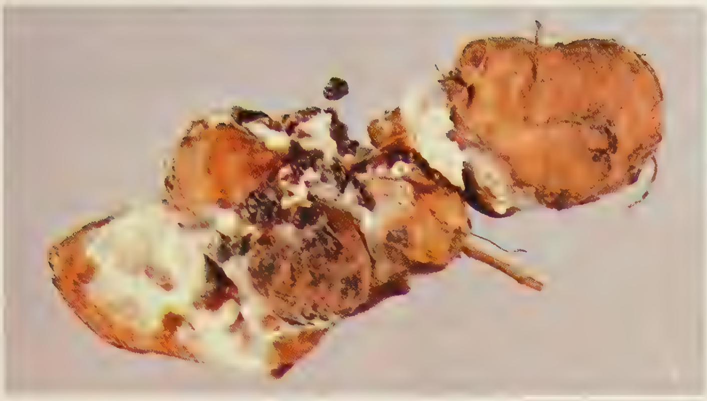
BLUE MOULD ROT is caused by species of *Penicillium*, which appear to be only weakly parasitic on Jerusalem artichokes. Infection occurs through wounds or via lesions made by other fungi, and decay is significant only at temperatures of 20°C and above (4).

FUSARIUM ROT may be caused by several species, for example *Fusarium oxysporum* (page 249) and *F. pallidoroseum* (page 34) (8). Rotting is rapid at 25°C but is largely held in check by refrigeration, unless soft-rot bacteria are present in association.

GREY MOULD ROT, caused by *Botrytis cinerea* (page 281), is an important storage rot in the USSR (2) and the northern USA (4). It is characterised by pale brown, slightly sunken lesions which, in a humid atmosphere, become covered with white mould giving rise to grey-brown spores (4). Internally the affected tissue is pale brown and soft. Injuries predispose tubers to infection, and rotting can occur even at low temperatures.

RHIZOCTONIA ROT is seen occasionally on Jerusalem artichokes in the southern USA (8) and has also been recorded in Malaysia. The causal fungus is *Thanatephorus cucumeris* (sclerotial state: *Rhizoctonia solani*) (page 307) and it incites a brown rot of the tubers.

RHIZOPUS ROT can cause severe decay (4). *Rhizopus stolonifer* makes substantial progress at temperatures between 6° and 20°C (4) while *R. tritici* is most active at temperatures above 20°C. Infected tissue is dark brown and soft, and in a humid atmosphere there may be copious growth of mould bearing characteristic spore-heads (page 81).



393 Watery soft rot

SCLEROTIUM ROT of Jerusalem artichokes has been recorded in the southern USA (4), Ghana and Malaysia (9). The causal fungus is *Corticium rolfsii* (sclerotial state: *Sclerotium rolfsii*) (page 255). Serious losses can occur in the field (8), through collar rot, wilt, root- and tuber-rot, and tubers appearing sound at harvest may later rot in store (9). The fungus produces a characteristic silky white mould, on which form abundant spherical resting bodies (sclerotia). Disease control in the field is difficult owing to the persistence of sclerotia in the soil and the expense of chemical treatment (8). Post-harvest rotting can be completely arrested by storage at low temperature or, if refrigeration is unavailable, by the use of a fungicide dip (9).

WATERY SOFT ROT is caused by species of *Sclerotinia*, responsible for the most important disease of Jerusalem artichokes grown in temperate countries. *S. minor* (page 212) has been recorded in the UK (5), France (3) and Chile, and *S. sclerotiorum* (page 47) in Canada (7), New Zealand (1), the UK, France (3) and northern and central USSR (2,6). The disease is especially serious in heavy acid clay soils where drainage is poor (2). Wet conditions permit 'germination' of sclerotia, leading to above-ground infection by ascospores of *S. sclerotiorum* (1,2) or ground-level infection by mould strands of *S. minor* (3). Infection progresses from the stem into the tubers (1); plants may wilt and die (5,7), or tubers may appear healthy at harvest but later rot in store (3). Control measures include improvement of drainage (2), destruction of weeds and prompt removal of diseased plants (6). It should be remembered that infected seed tubers can transmit the disease to new areas (1).

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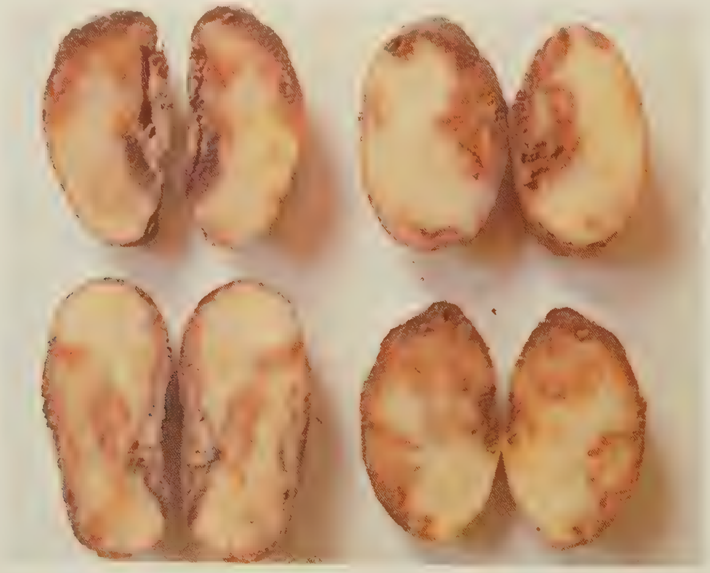
Potatoes

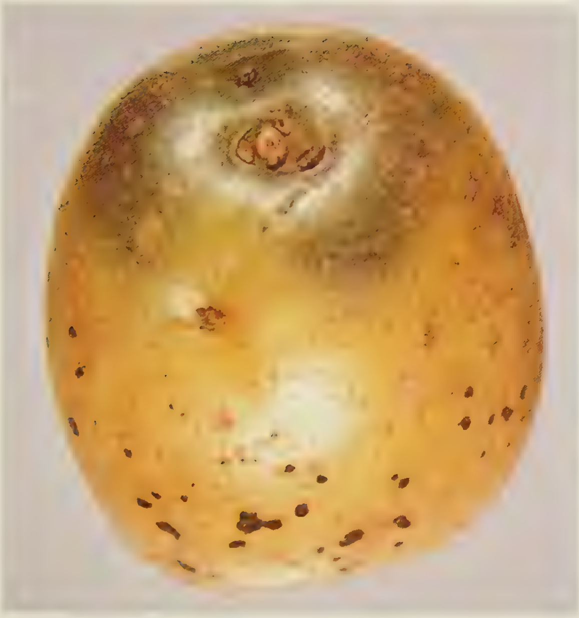
The **potato**, now one of the world's most important food crops, had its origins in the high Andes of Peru, where it has been under cultivation for perhaps seven thousand years (1). Wild potato tubers are generally bitter-tasting and potentially toxic, and the first stage in the evolution of the crop must have been the recognition and selection of less bitter types (3). The modern potato is *Solanum tuberosum* L., of which two subspecies have become differentiated, ssp. *andigena* the typical Andean form still grown in South America, and ssp. *tuberosum*, developed as a result of selection under long-day conditions, as in coastal Chile and, during the last few centuries, in Europe (1). The date of the potato's introduction into Europe is believed to have been about 1580. Interpretation of records is difficult owing to confusion with other South and Central American roots and tubers collected during the voyages of discovery. The unrelated lowland root crop, now known as the sweet potato *Ipomoea batatas*, had been called 'batata' or 'patata' in its Caribbean homeland, and this word, which became 'potato' in English, was at first used as a general term for all similar-looking foreign roots and tubers. Thus it was that *Solanum tuberosum*, eventually becoming pre-eminent in Europe, usurped the name 'potato' (1). *Ipomoea* was given the name 'sweet potato' or (preferably, in view of the lack of relationship) 'sweetpotato' (q.v.). In countries where both crops are grown, *Solanum* is sometimes distinguished by the epithet 'white' or 'Irish' potato.

The Irish potato has had a profound influence on the course of history. Despite being in several ways inferior to grain, the introduced potato was soon to become the staple food of Ireland's peasantry, most of whom were so poor that they could afford little else. This dependence on a single perishable foodstuff carried grave risks. In 1845 and 1846 the crop was devastated by blight (q.v.), and famine ensued on an unprecedented scale. A million died and a greater number emigrated to North America and elsewhere. The social and political repercussions of 'The Great Hunger' are still felt (8). Today the potato is grown in most countries of the world except for the humid lowland tropics, but it is of particular importance in northern and central Europe and in the Soviet Union. World production is approximately 300 million tonnes, the top ten producers being the USSR, Poland, China, the USA, India, Romania, the Netherlands, France and Germany. A significant proportion goes for stockfeed, but most is for human consumption; in several countries the potato is eaten daily, in sufficient quantity to contribute significantly towards the dietary requirement of vitamin C (3). Potatoes may be boiled, baked, roasted or fried. Cylindrical slices, fried in oil and eaten hot, are known as chips in the UK and as French fries in North America. Wafer-thin slices, fried in oil, drained and then packaged for eating out of hand, are called crisps in the UK and, confusingly, chips in North America (9). There are important industries for crisps, frozen raw chips and canned new potatoes, as well as for the production of potato starch and alcohol (schnapps).

Botanically the plant is a dicotyledon, in the same family (Solanaceae) as tomatoes, peppers and eggplants (q.v.). The potato plant occasionally produces berries, the seeds of which (true seed) are used in breeding (3,4). The tubers develop from underground shoots (stolons), by which they remain attached to



394 Blight

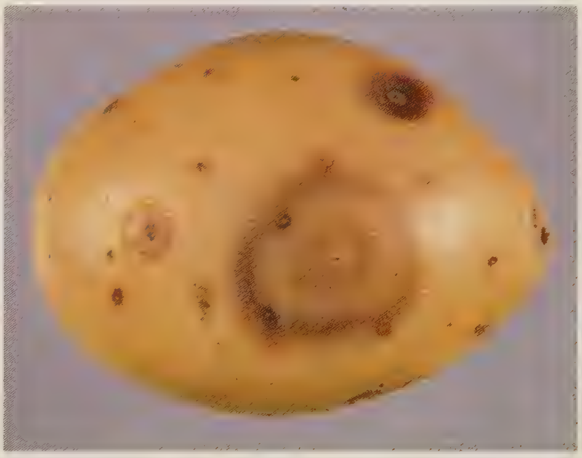


395 Field greening and black scurf

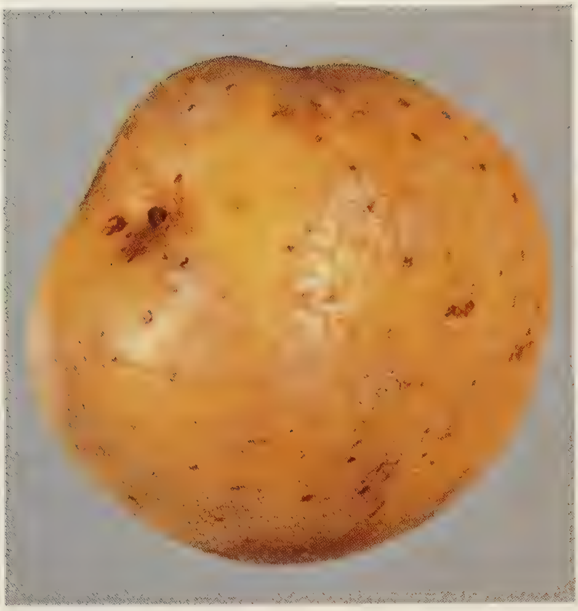
the mother plant. Commercial propagation is by means of whole tubers (seed potatoes) or parts of tuber cut to include at least one bud or eye (seed pieces). Much importance is attached to the production of healthy certified seed stocks, raised in chosen localities and under conditions which preclude the development of certain viral, bacterial and fungal diseases (3,5,6,10). In order to ensure that seed tubers will remain sound and produce a healthy crop, they are subjected to a fungicide treatment by dipping, fogging or, increasingly, spraying. Protecting potatoes at this stage, compared with treating the harvested ware crop, is advantageous in two ways; firstly a smaller quantity of tubers is involved, and secondly there is not the question of chemical residues in tubers which are to be consumed.

If potatoes are harvested when the haulm (stem and foliage) is still green and vigorous, the tubers are immature: they have thin delicate skins and can be sold as 'new potatoes' or 'scrapers', which fetch a premium price in the market. However, most ware crops are left in the ground until the haulm has died down or been deliberately killed off with chemicals; mature tubers have a set skin which cannot be removed except by peeling. Harvesting and grading can result in significant physical damage in the form of cuts, abrasions and deep bruising (2,7,10). Not only does this detract from tuber quality (leading to downgrading in the market or rejection by processors), but it also increases moisture loss and facilitates invasion by fungi and bacteria which are ever-present in the adhering soil (5,6,10). Damage can be minimised by careful adjustment of machinery and scrupulous training of personnel.

The storage of potatoes has been the subject of intensive study and experiment (1,3,7,8). Following on from the use of clamps, pits and barns, efforts were put into the design and construction of specialised potato stores, some to house the crop in bulk, others to accommodate pallet boxes. In many temperate countries the climate permits winter storage of potatoes in buildings ventilated with ambient air; to guard against freezing, a thermostat determines whether the vents admit fresh or recirculated air. Alternatively stores may be equipped with refrigeration machinery, and there have been sophisticated developments in micro-processor control of temperature and humidity (4,7). If, as often happens in northern Europe, the crop is lifted from wet soil, the first necessity is the elimination of surface moisture by appropriate ventilation; the persistence of free water on tubers and soil could otherwise lead to microbial attack. This 'drying' process can take several hours. Subsequently humidification of the atmosphere may be needed, especially during long-term storage, to prevent excessive weight loss through evaporation of moisture from tuber tissue. A high humidity, coupled with moderate temperature, has usually been considered essential during the first phase of storage, that of 'curing' the tubers. Such conditions favour the healing of wounds and the development of a barrier against microbial attack. The same conditions also favour the micro-organisms, however, and in situations where there is considered to be a high risk of bacterial soft rot, 'dry curing' may be preferred; this involves rather lower humidities, and makes the curing process longer but safer. Temperature is very influential; at optimal humidity, complete curing may take only 3 to 6 days at 20°C, 1 to 2 weeks at 10°C and 3 to 6 weeks at 5°C. At lower temperature, wound healing occurs very slowly or not at all, and during long-term storage, infection and decay can lead to heavy losses.



396 Fungal and bacterial infection via lenticels

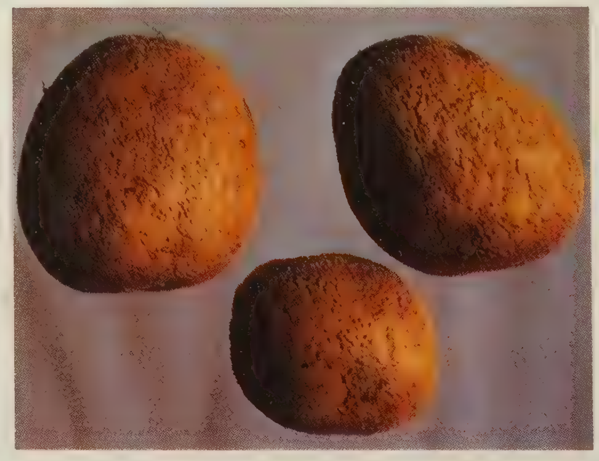


397 Silver scurf

The optimal storage environment for potatoes depends on their intended use. Seed potatoes are best stored at 2° to 4°C and subsequently exposed to warmth and light for 'chitting', i.e. the development of small sturdy sprouts prior to planting. Ware potatoes, on the other hand, need to be kept in complete darkness, since exposure to light causes greening (q.v.) and toxin-formation which would render them unfit for consumption. Optimal storage temperatures are 5° to 6°C for fresh market potatoes, 6° to 8°C for chipping potatoes and 7° to 10°C for crisping potatoes. The significance of temperature is the effect it has on the starch in the tuber (1). During storage there is a tendency for starch to be converted into sugars, and this process is accelerated at low temperature. Excessive sugar content is undesirable in potatoes for the fresh market and especially so in potatoes intended for chipping and crisping, since it results in an unattractive dark brown colour in the cooked product. On the other hand, temperatures above 5°C permit sprouting and decay. The optimal storage temperature is thus a compromise based on specific requirements. Sprouting may be held in check either by means of a chemical spray applied pre-harvest or by the use of a fumigant in store after curing is complete. Irradiation is also effective in suppressing sprouting, though its effects on cooking quality must also be taken into account. Low temperature sweetening may be reversed by holding affected batches at 20°C for about two weeks; on the other hand, senescent sweetening (resulting simply from overlong storage) is irreversible. Cultivars differ markedly in their intrinsic properties and in their response to storage regime.

When potatoes are brought out of store, there are renewed opportunities for damage, especially during grading and sorting (7). Tubers are particularly susceptible to bruising if cold, and it is therefore advantageous to warm them to about 15°C before removal from store. After all the preparatory handling operations, potatoes may be despatched in bulk to a processor or packaged for the fresh market. Sacks are commonly used at wholesale level, while perforated polyethylene makes a convenient supermarket pre-pack. Consumer demand for washed, blemish-free tubers has led to increased efforts to control disfiguring diseases such as silver scurf, black scurf and black dot (q.v.). Current practices also necessitate precautions against bacterial soft rot and greening (q.v.).

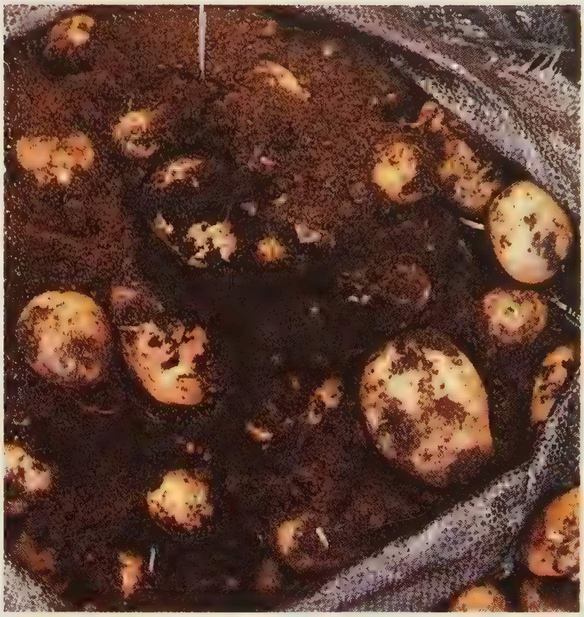
Such is the demand for 'new potatoes' in Europe that an important trade has developed with Egypt, Cyprus, Greece and the Canary Islands which, by virtue of a warm climate, can supply an early spring crop. Export consignments were traditionally packed in jute sacks, or occasionally cartons, and carried in ventilated ships at ambient temperature. The Egyptian trade in particular was characterised by the use of sacks lined with a perforated black polyethylene bag and containing moistened peat-moss. The primary function of the damp peat-moss was to protect the freshly harvested tubers and to prevent skin-set which would detract from their market value as 'new scraper potatoes'. An unintentional but highly relevant benefit conferred by the peat-moss is the cooling effect resulting from evaporation of moisture. The disadvantage of the peat-moss is the risk of free water being expelled from clumps of peat-moss subjected to pressure in the stow. Inadequate stowage and ventilation can lead to complete or partial loss of cargo through self-heating and soft rot. In a ventilated ship it is essential to stow the sacks in blocks separated by air channels, in a manner which ensures that air can penetrate the stow.



398 Skin spot

Wooden planks (dunnage), placed within and beneath the stow, help to maintain the air channels and prevent collapse of the stow during the pitching and tossing of the vessel. When jute sacks were superseded by woven polypropylene sacks, increased care became necessary to prevent the slippery packages from moving unduly in passage.

In recent years there has been increased use of refrigerated vessels for spring and summer shipments from Egypt. Prompt and thorough cooling to 4° or 5°C is effective in suppressing sprouting and preventing bacterial soft rot. Refrigerated carriage of potatoes has one potential hazard which is not always appreciated. If a cold cargo is discharged into a warm humid atmosphere, there is a grave risk of condensation followed by anaerobiosis (page 302), followed in due course by massive bacterial soft rot. It is therefore prudent (starting 2 or 3 days before arrival) to allow the cargo to warm to a temperature above the expected atmospheric dew-point. Even in the absence of condensation there remains a possibility of losses from bacterial soft rot if potatoes are held in warm, ill-ventilated conditions after discharge from the vessel; rotting is facilitated by the high moisture content of the peat-moss, since little evaporation will have occurred in the confines of a refrigerated hold.



399 Bacterial soft rot

Depending on the Plant Health Regulations of the importing country, a potato cargo may be impounded if it is found to be harbouring certain non-indigenous pests and diseases. In the UK, for example, there are rules (of varying strictness, depending on the perceived degree of risk to home-grown crops) relating to Colorado beetle, potato tuber moth, ring rot (q.v.) and brown rot (q.v.). In order to ensure that cargoes are free (or, if applicable, substantially free) from relevant pests and diseases, it is vital that effective control measures are carried out in the country of origin.

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2 CAPPELLINI R.A., CEPONIS M.J., WELLS J.M. & LIGHTNER G.W. (1984) Disorders in potato shipments to the New York market, 1972–1980. *Plant Disease* **68,** 1018–1020.

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See also:

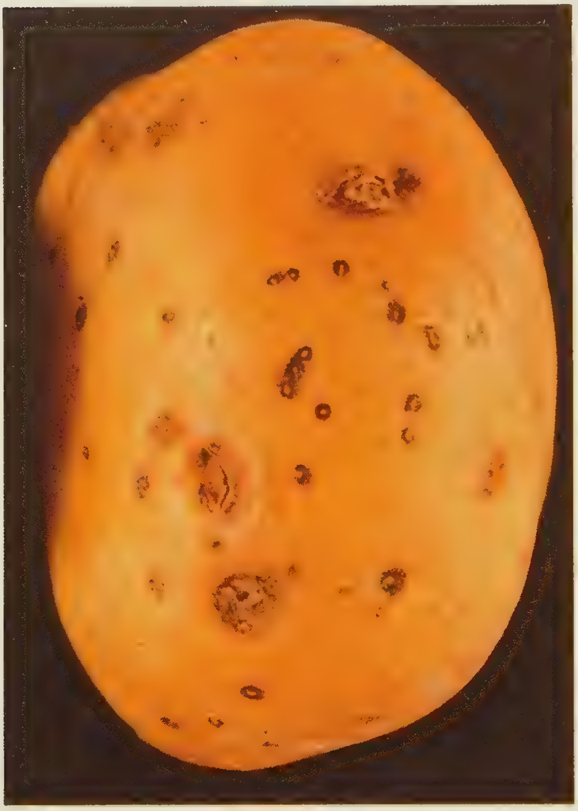
CARGILL B.F., BROOK R.C. & FORBUSH T.D. (Eds.) (1989) *Potato storage, technology and practice. Proceedings of an international symposium, Michigan State University, East Lansing, MI, USA, 1985.* St Joseph, Michigan, USA: American Society of Agricultural Engineers, 340 pp illus.

GUNN J.S. (Ed.) (1990) *Crop protection handbook – potatoes.* Farnham, Surrey: British Crop Protection Council, 192 pp.

ALTERNARIA ROT of potatoes caused by

Alternaria alternata **(Fr.) Keissler**
Alternaria solani **Sorauer**

400



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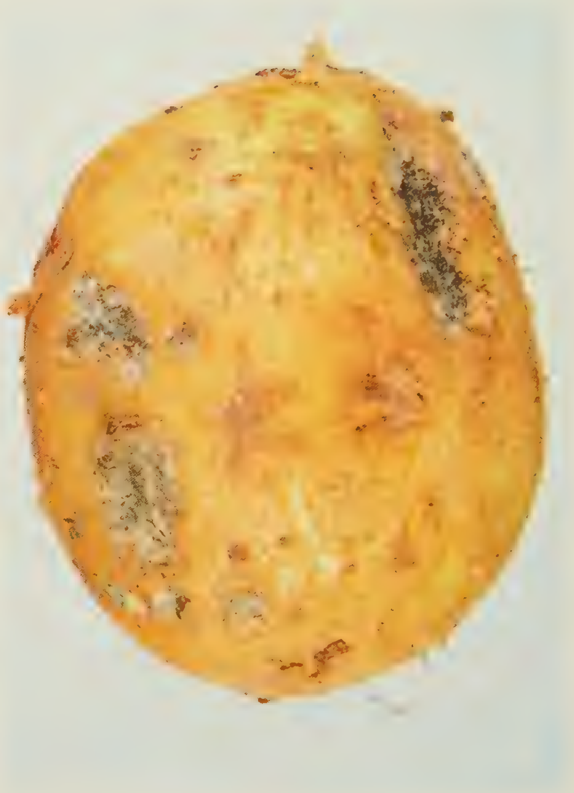
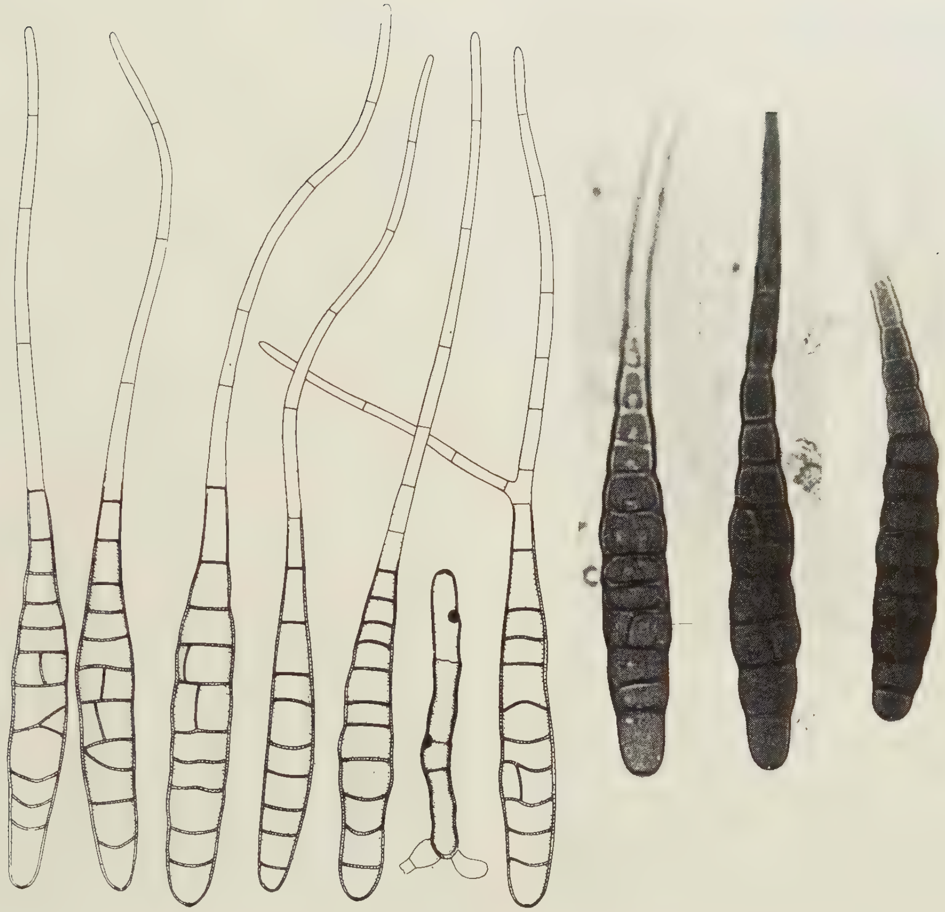


OCCURRENCE Both fungi are widespread in temperate and tropical countries. *A. alternata* has recently been recorded on stored potatoes in Israel (1). Tuber-rotting by *A. solani* (the cause of 'early blight' of foliage) has been reported from Germany, the Netherlands (3), Australia (8) and the USA (2,7).

SYMPTOMS *A. alternata* can cause numerous dark shiny sunken lesions, and the disease has been named **black pit** (1). The lesions induced by *A. solani* are also dark and depressed, sometimes with slightly raised purplish black borders. They may be small and circular (2) or extensive and irregular in shape (8); infected tissue is firm, leathery and sharply delimited from the healthy flesh beneath (2).

BIOLOGY The fungi survive in the soil and on crop debris (6), their spores (conidia) being dispersed by wind. Infection of foliage is especially likely during periods of alternating dry and wet conditions (5,8), dew being as significant as rain or irrigation water (1,9). Plants under stress are predisposed to infection, and there may be epidemic development of blight on the foliage (8,9). Even if leaf infection is slight, there is a risk that tubers may become infected at harvest time, especially if they are immature (2). During harvesting and handling, the tubers become contaminated with spores and invasion can occur, occasionally via the natural openings (lenticels) (8) but much more commonly through injuries such as abrasions (1,8). Both incidence and severity of storage rot are much greater in mechanically harvested tubers than in those lifted by hand (1).

CONTROL Disease forecasting programmes have been devised (5) and, after a series of investigations on control measures in the USA (Colorado), it has been recommended, where appropriate, that fungicide be applied via the sprinkler irrigation system. Care should be taken to minimise injury during harvesting, and pre-storage conditions should be conducive to efficient curing (page 298), since the risk of infection is greatest while wounds remain unhealed (7,8). A post-harvest fungicide treatment may be applied to tubers for added protection (1,4). Some potato cultivars are partially resistant to early blight (3,5,10).



402 *Alternaria solani* (from CMI Descr. No. 475, Ellis & Gibson 1975) Conidia and conidiophores × 500

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1 DROBY S., PRUSKY D., DINOOR A. & BARKAI-GOLAN R. (1984) *Alternaria alternata:* a new pathogen on stored potatoes. *Plant Disease* **68,** 160–161 illus.

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7 NNODU E.C., HARRISON M.D. & WORKMAN M. (1982) The effect of storage environment on the infection of potato tubers by *Alternaria solani. American Potato Journal* **59,** 313–325.

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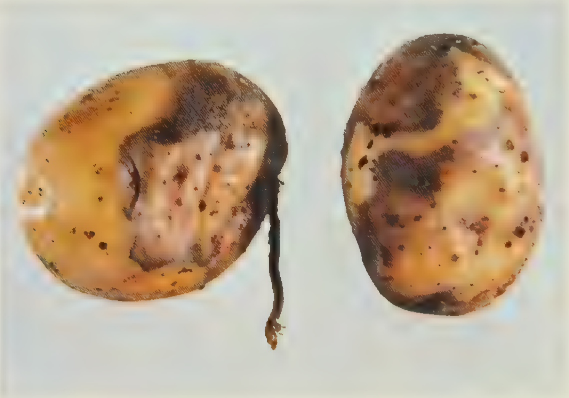
BACTERIAL SOFT ROT of potatoes caused by *Erwinia* ssp.

403



OCCURRENCE Bacterial soft rot, and the associated field disease known as 'blackleg', can cause heavy losses wherever potatoes are grown. *Erwinia carotovora* ssp. *atroseptica* (van Hall) Dye is of particular importance on potatoes in temperate countries such as the UK (3,5,7); in tropical regions it is confined to the cool highlands (6). *Erwinia carotovora* ssp. *carotovora* (page 271) exists in all climatic zones and attacks many other crops besides potatoes (8). *Erwinia chrysanthemi* pv. *chrysanthemi* Burkh., McFadden & Dimock likewise has a wide host-range; it is found chiefly in the lowland tropics (4,6) and in other warm areas such as parts of Australia (2).

SYMPTOMS Lesions centred on natural openings (lenticels) in the tuber are typically circular, while those associated with injuries are more likely to be irregular in shape. Infected areas may also be found at the heel end (8). Soft-rotted tissue is characteristically creamy at first, and later black. Under conditions favourable to bacterial growth, rotting in a mass of potatoes can be extremely rapid. There is a characteristic rise in temperature consequent upon enhanced respiratory activity of the infected tubers coupled with the heat of respiration of the bacteria. In advanced stages of decay the development of secondary organisms (such as *Clostridium* sp.) results in a foul odour (10), and there is often much exudation of fluid. Alternatively, a non-progressive 'bacterial hard rot' (q.v.) may develop if infection is arrested at an early stage by storage in a dry atmosphere (1).

BIOLOGY The bacteria may be present in soil (2), in irrigation water, in crop debris and in tubers which escaped harvest (8). The most common source of infection, however, is the seed tuber. An infected tuber may decay without producing a plant, or it may give rise to a plant with 'blackleg', characterised by a dark lesion at the base of the stem (3,5,7). Daughter tubers may be invaded directly via the point of attachment (stolon) (**403**). Furthermore, when rain or irrigation water percolates through the soil, bacteria (from soil, seed tubers or stem lesions) are carried to the surface of the progeny tubers (5) and lodge in the lenticels and growth cracks. Contamination is common, but infection and rotting occur only in certain circumstances (10). Fungal lesions or unhealed wounds provide a means of entry for the bacteria. Invasion via lenticels becomes possible if normal respiration of the tuber is disrupted by an insufficient supply of oxygen (anaerobiosis). This can follow very high rates of respiration induced by solar heat (when the increased demand for oxygen cannot be met), or it can follow inhibited respiration where gaseous exchange is restricted. The latter may be caused by a lack of ventilation, or by the presence of a film of water over all or most of the tuber surface (10). Under such conditions, lenticels can be breached within 2½ hours at 22°C or within 6½ hours at 10°C. Thus, if harvested potatoes are wetted by rain or by condensation, subsequent losses are likely to be catastrophic. Another mode of infection is 'infiltration' – if harvested tubers are immersed in deep washing-tanks, even for only a few minutes, there is a risk of water and bacteria being forced in through the lenticels by hydrostatic pressure (1). Rapid soft rotting ensues.

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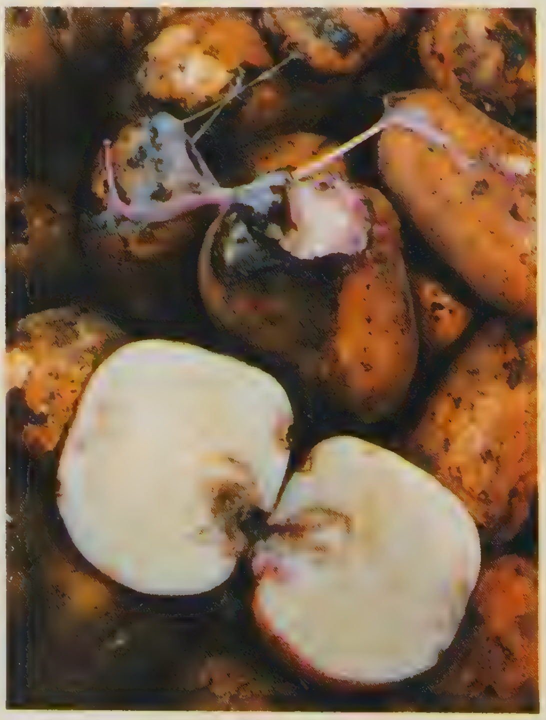

CONTROL It is desirable to plant clean seed tubers (derived from stem-cuttings) or disinfected seed tubers. It may be appropriate to apply a fungicide seed-dressing against fusarium rot (q.v.) which might otherwise predispose tubers to bacterial decay. Alternatively they can be subjected to a heat treatment which kills bacteria and other organisms (7). There is a high risk of recontamination in some soils, however, (2,3,6). Fields should be well-drained and irrigation carefully controlled, to reduce the likelihood of contaminated tubers (whether seed or progeny) becoming infected via the lenticels (2,8). Cull-piles of potatoes from the previous season should not be left to rot, since insects may transmit bacteria to the growing crop (3). If it is feasible, plants showing blackleg lesions are best removed, to prevent further spread of bacteria by rainsplash, insects or farm machinery (3,5). Tubers should not be harvested in very cold or in very hot weather, nor be handled roughly, since frost, heat or mechanical damage increases susceptibility. Frequent disinfection of harvesting and grading machinery is important, since bacterial slime from a single rotting tuber can contaminate many healthy potatoes (8). Applying bactericidal chemicals to the tubers has generally been ineffective (10), although a few substances show promise. Correct storage practices are essential (page 297), and errors can result in massive losses. An early warning system has been devised to detect the onset of bacterial soft rot by monitoring the volatile products given off by a stack of potatoes (9); a rise in temperature is a further indication. Bagged potatoes transported by ship must be properly stowed to permit efficient airflow through the cargo, and precautions should be taken to prevent condensation of moisture on or within the stow (page 299). Peat moss used in packaging should be damp rather than wet, and uniformly distributed within the bag, in order to lessen the risk of free drainage of water. If potatoes are to be washed for marketing, deep tanks and prolonged immersion should be avoided (1). Water must be changed frequently; chlorine added as a disinfectant is rapidly inactivated in dirty water. Washed tubers should be allowed to dry promptly and, if polyethylene packaging is used, the bags should be perforated to permit ventilation. Work is in progress to investigate mechanisms of resistance (4) and to breed resistant potato cultivars (3).

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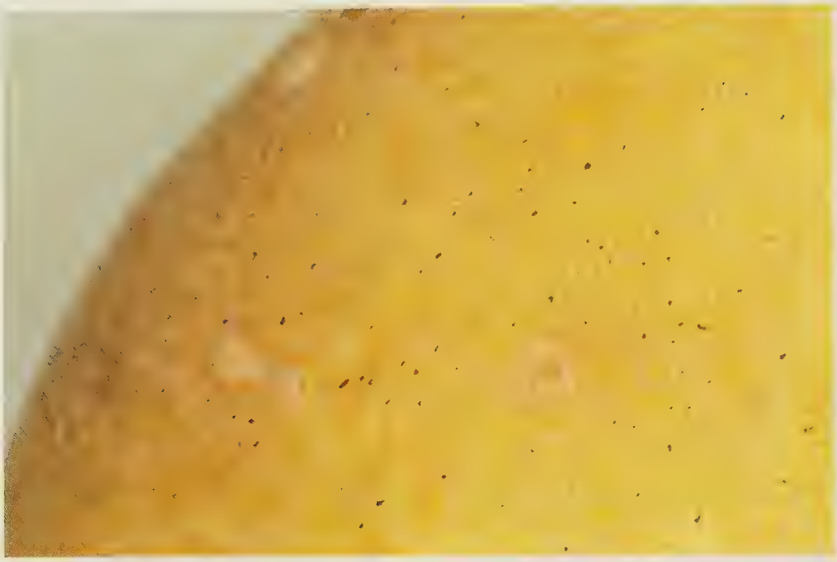
See also:

KLOPMEYER M.J. & KELMAN A. (1988) Use of monoclonal antibodies specific for pectate lyase as serological probes in the identification of soft rot *Erwinia* ssp. *Phytopathology* **78,** 1430–1434.

BLACK DOT of potatoes caused by ***Colletotrichum coccodes*** **(Wallr.) Hughes**

406



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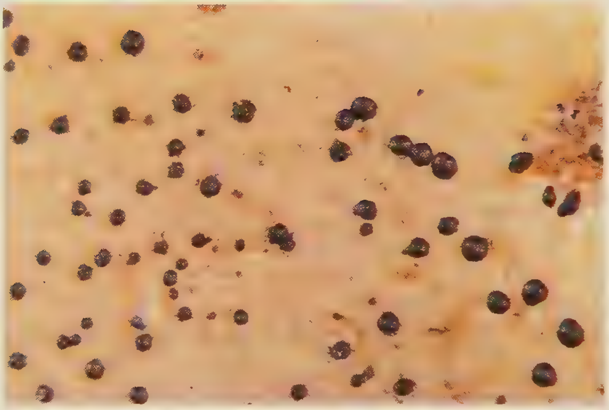
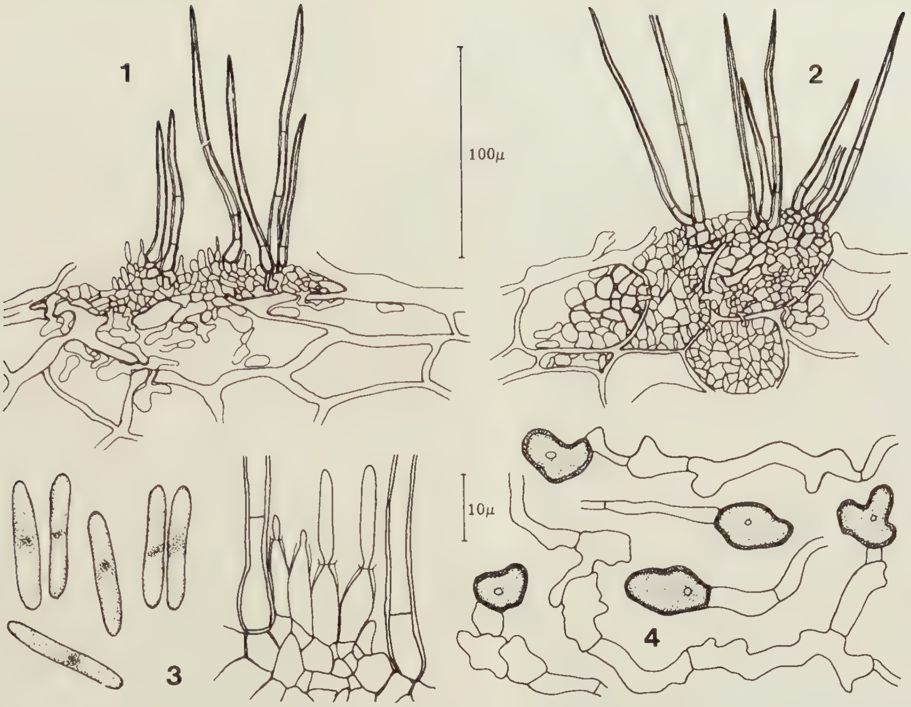


OCCURRENCE The fungus occurs in most potato-growing countries, for example the UK (1), France (4), Germany (8,10), the Netherlands (9), the USSR (3), the USA (2,6,7), India and Australia (5). It is more prevalent in wet than in dry seasons (10). Consumer demand for unblemished tubers has brought this minor disease to prominence in recent years (6,8). Symptoms are especially noticeable on washed tubers of coloured cultivars.

SYMPTOMS Minute black bodies, barely visible to the naked eye, may occur on any part of the tuber, and the surrounding skin becomes dry and discoloured (9). Symptoms may resemble those of silver scurf (q.v.), with which black dot is often confused; microscopic examination serves to distinguish the two fungi, which sometimes occur together (6). When the heel end of the tuber is infected, the fungus can cause a dark sunken rot (5).

BIOLOGY The black bodies are minute sclerotia, which characteristically possess dark 'bristles' (setae) and give rise to the asexual stage of the fungus (setose acervuli with conidia), the spore masses being pinkish in colour (2,6,10). The organism is transmitted on seed tubers (5,7); it also persists in soil and on infected crop debris and weeds (4,8). Infection of growing plants is especially likely in the presence of other root- and stem-invaders such as *Rhizoctonia solani* (q.v.) (10). Field symptoms include yellowing and wilting of foliage (4,5), and tubers may show blemishes at harvest time (3). The later the harvest date, the higher the incidence of black dot (9). Disfigurement is exacerbated if the storage temperature falls to −1°C, especially if the atmosphere is humid (9). Infected tubers are particularly prone to weight loss by increased evaporation of moisture from the damaged areas (6).



408 *Colletotrichum coccodes* (from CMI Descr. No. 131, Mordue 1967) 1 acervulus 2 sclerotium 3 conidia and conidiophores 4 appressoria and hyphae

CONTROL Infected crop debris should be removed and burned, and weeds kept under control (5). Crop rotation can be helpful; it should be borne in mind that other susceptible hosts include tomato, pepper and eggplant (4). It is desirable to obtain clean seed potatoes, since fungicide treatment of infected tubers is not always effective (1). Potatoes harvested from wet soil should be surface-dried at commencement of storage (page 297).

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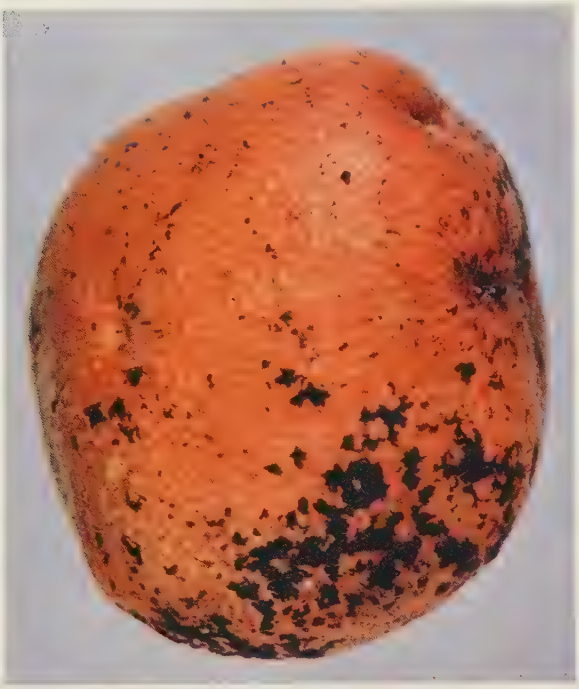
10 SCHMIEDEKNECHT M. (1956) Untersuchung des Parasitismus von *Colletotrichum atramentarium* (B. & Br.) Taub. an Kartoffelstauden (*Solanum tuberosum* L.). *Phytopathologische Zeitschrift* **26,** 1–30 illus.

BLACK SCURF of potatoes caused by
Thanatephorus cucumeris **(Frank) Donk**
Sclerotial state: *Rhizoctonia solani* Kühn

409



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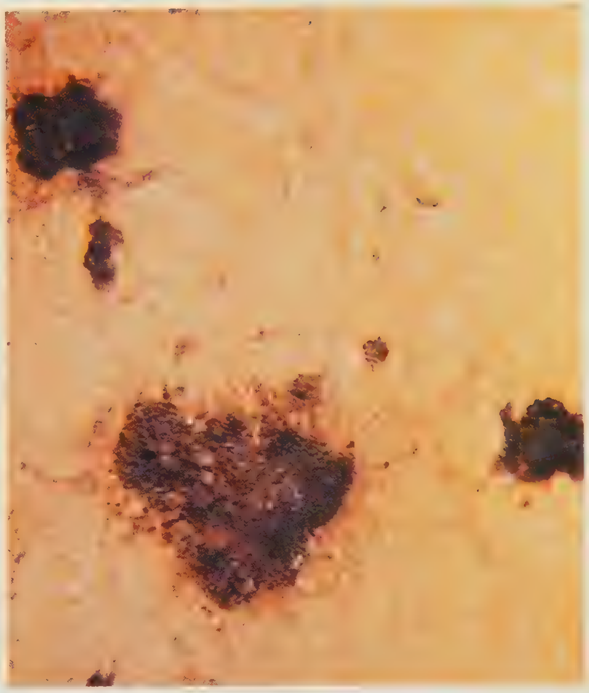
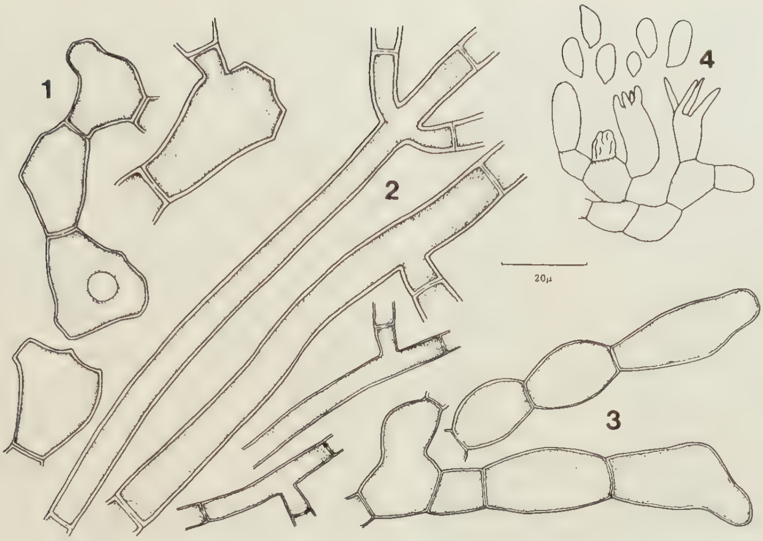


OCCURRENCE Black scurf, and associated stem canker, occur in most potato-growing countries, for example Canada (5), the USA (2,6), the UK (1,3,7, 10), the Netherlands (4,8) and India (9). Although the fungus occasionally causes tuber decay (10), it is of greater importance as an unsightly market blemish.

SYMPTOMS The most noticeable symptom is the presence of dark brown to black bodies, up to several mm across and closely adpressed to the tuber surface (3). These structures (sclerotia) may be mistaken for lumps of soil, but they are not removed by washing. Examination with a hand lens may reveal a sparse growth of dark reddish-brown fungal strands spreading out from the sclerotia. Occasionally the fungus can cause a rot, especially at the heel end, and sclerotia may then be formed within the tissue (10).

BIOLOGY The fungus survives as sclerotia in the soil and as mould strands in crop debris; it can be transmitted to new areas on infected seed tubers (1,5,6). Sprouts developing from infected seed tubers are liable to be invaded by mould strands growing out from tuber-borne sclerotia (1,5). The killing of shoot-tips results in delayed emergence and consequent retardation in plant development (7). The stem canker phase of the disease is characterised by dark, spreading lesions on the stem (7). It is at this stage that the fungus may produce the sexual state (a hymenium giving rise to basidiospores), visible as a thick greyish-white 'mat' enveloping the stem (2,9). Spores are dispersed by air currents and can cause new infections of aerial plant parts (9). At the same time, underground parts (roots, stolons and tubers) may be invaded by mould strands originating from soil-borne sclerotia (5). Formation of sclerotia on the daughter tubers is initiated at any time, but maximal development occurs after the haulms have been destroyed prior to harvest (this is probably because physiological changes in the tubers increase their susceptibility) (3,6). Both incidence and severity of black scurf may increase during storage (1,3), depending on the temperature and relative humidity in the store and the amount of soil and debris in the harvested crop (10). If the fungus is able to penetrate wounds it may also cause a rot, and this is especially likely in a moist storage environment (10).

CONTROL Potato cultivars differ greatly in susceptibility to black scurf. Crop rotation helps to reduce the level of soil infestation (5). Seed tubers should be treated with a fungicide before or during planting (7), since even unblemished tubers may be carrying the fungus (5). Since few fungicides are completely effective against sclerotia, there have been attempts



411 *Thanatephorus cucumeris* (from CMI Descr. No. 406, Mordue 1974)
1 sclerotial cells 2 hyphae 3 monilioid cells 4 basidia and basidiospores

at biological control, which is most effective when combined with chemical treatment of the soil (8). Close examination of the eyes of developing progeny tubers can give an indication of the level of infection (1) and, if other considerations permit, it may be prudent to harvest the crop at an early date, within 2 to 3 weeks of haulm destruction (3,6). Flexibility in harvest date can be achieved if, as a supplement to chemical haulm destruction, the roots are cut off (4). Not only does this accelerate skin-hardening (making earlier harvesting feasible) but it also appears to delay the development of black scurf, making later harvesting a possible alternative (4).

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3 CHAND T. & LOGAN C. (1984) Post-harvest development of *Rhizoctonia solani* and its penetration of potato tubers in Northern Ireland. *Transactions of the British Mycological Society* **82,** 615–619 illus.

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6 GUDMESTAD N.C., ZINK R.T. & HUGUELET J.E. (1979) The effect of harvest date and tuber-borne sclerotia on the severity of *Rhizoctonia* disease of potato. *American Potato Journal* **56,** 35–41.

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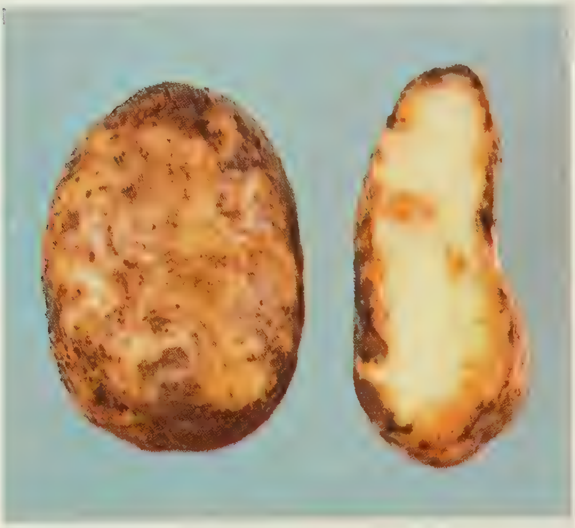
8 JAGER G. & VELVIS H. (1986) Biological control of *Rhizoctonia solani* on potatoes by antagonists. 5. The effectiveness of three isolates of *Verticillium biguttatum* as inoculum for seed tubers and of a soil treatment with a low dosage of pencycuron. *Netherlands Journal of Plant Pathology* **92,** 231–238.

9 KISHAN SINGH, MISRA S.R. & CHAUBEY R.D. (1984) Occurrence of *Thanatephorus cucumeris* (Fr.) Donk on potato in India. *Indian Journal of Plant Pathology* **2,** 132–134 illus.

10 SPENCER D. & FOX R.A. (1979) Post–harvest development of *Rhizoctonia solani* Kühn on potato tubers. *Potato Research* **22,** 41–47.

BLIGHT of potatoes caused by ***Phytophthora infestans*** **(Mont.) de Bary**

412

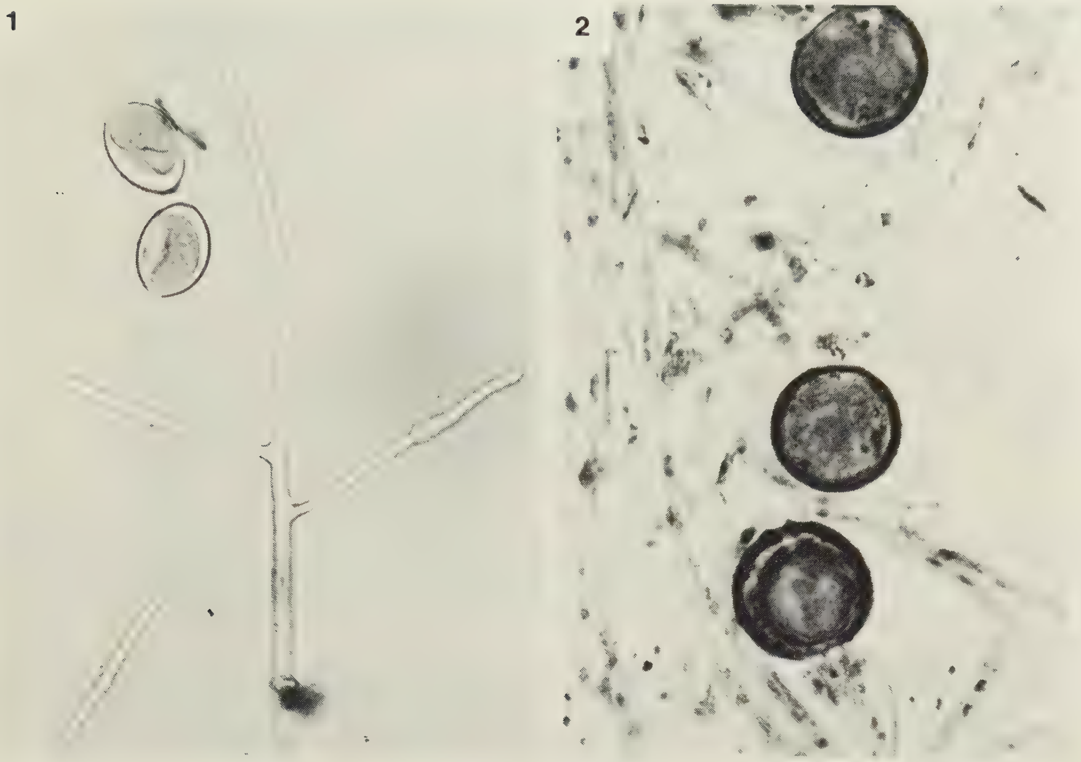


OCCURRENCE The potato blight fungus has a world-wide distribution (CMI Map 109). It was blight which devastated the potato crop in Ireland in the 1840s, indirectly precipitating famine, death and migration. Outbreaks of the disease tend to be frequent in maritime areas such as the British Isles (4) and Japan (8) and at high altitudes in tropical and sub-tropical countries where there is much cloud and mountain mist (2). The disease also occurs in arid areas under irrigation, such as parts of Israel (7) and the USA (3). Where there is a true continental climate, such as in the USSR and Poland (6), blight occurs less often but may nevertheless occasionally reach epidemic proportions if conditions permit (2). To distinguish it from 'early blight' (caused by *Alternaria solani*, q.v.), the disease is often called 'late blight' (3,10).

SYMPTOMS External lesions on infected tubers are irregular in shape, and tend to be dark and slightly sunken. In a humid atmosphere tufts of fine white mould may be present at eyes or lenticels. If an infected tuber is cut in half, a reddish-brown 'marbling' of the flesh may be apparent in the region just beneath the skin. Later the entire tuber rots, often becoming soft and slimy as a result of invasion by bacteria. The blight fungus occasionally produces the sexual state (thick-walled oospores), first reported from Mexico (5) and subsequently found in the UK and Switzerland. It is the asexual state, however, which is of primary importance (2).

BIOLOGY In addition to being transmitted in seed potatoes (3), the fungus survives unfavourable periods in tubers which have escaped harvest or been left in cull-piles (10). When an infected tuber sprouts, the fungus may grow up into the shoot and, in mild humid conditions, produce numerous asexual spores (sporangia). Liberation of these spores is aided by changes in humidity, and dispersal is by wind and water (2). If foliage remains wet for some time, whether from dew (4), rain (8) or irrigation water (3), spores which have landed on the leaf surface are able to germinate. At temperatures around 20°C germination is direct (each sporangium producing a germ-tube), while at temperatures nearer 10°C it is indirect (each sporangium giving rise to several zoospores). If free water persists, penetration of healthy leaf tissue occurs. Under optimal conditions germination and infection can occur within 3 to 5 hours and, if conditions remain favourable, the process may be repeated many times, resulting in rapid spread within the crop (2). Leaf lesions tend to be predominantly at the tip, base or edge of leaflets, and appear as dark brown blotches. If humid conditions continue, a fine white spore-bearing mould develops just ahead of the advancing edge of the lesion. Spores are also produced at the edges of stem lesions (4). As water drips off the foliage or runs down the stem, it carries spores into the soil and perhaps into contact with developing tubers (8). Penetration of tubers is generally through eyes, lenticels, growth cracks or wounds, although occasionally the fungus invades via the point of attachment to the plant (the stolon) (4). Infection of tubers may occur before harvest if soil moisture is high enough (4) or after harvest, following contamination (during lifting of tubers) by spores from partially blighted foliage (3). Tubers appearing healthy at harvest time may later rot in store (3).

CONTROL It is essential to plant clean seed (3). Field hygiene is also important; all cull piles and volunteer plants should be removed and destroyed (10), since a single diseased shoot per square km can be sufficient to start an epidemic (3). Forecasting systems, based on combinations of temperature with relative humidity or rainfall, have been devised for various parts of the world (9,10). Taken in conjunction with field surveys, weather monitoring permits optimal timing of fungicide sprays (9,10). Because of the capacity of the fungus to evolve new strains, it is preferable to use a combination of fungicides with differing modes of action (7). Some potato cultivars are partially resistant to blight and require fewer spray applications than do susceptible cultivars (9). Tubers can be protected from contact with spores by 'earthing up' plants during the growing season, and by ensuring that blighted foliage is



413 *Phytophthora infestans* (from CMI Descr. No. 838, Stamps 1985) 1 sporangiophore and sporangia 2 oogonia with amphigynous antheridia and oospores × 500

destroyed a full 2 weeks before harvest (3). Cool storage and efficient ventilation serve to minimise development of blight in store, but some cultivars become increasingly susceptible during the storage period (1). If infected potatoes are shipped at ambient temperature, blight can develop during the voyage.

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3 EASTON G.D. (1982) Late blight of potatoes and prediction of epidemics in arid central Washington State. *Plant Disease* **66,** 452–455.

4 LAPWOOD D.H. (1977) Factors affecting the field infection of potato tubers of different cultivars by blight (*Phytophthora infestans*). *Annals of Applied Biology* **85,** 23–42.

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8 SATO N. (1980) Sources of inoculum and sites of infection of potato tubers by *Phytophthora infestans* in soil. *Annals of the Phytopathological Society of Japan* **46,** 231–240.

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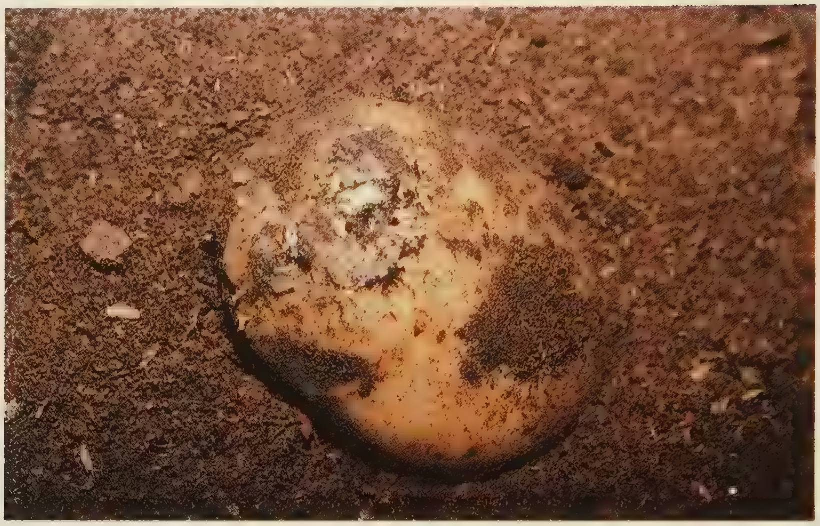
BROWN ROT of potatoes caused by
Pseudomonas solanacearum **E.F. Smith**

414



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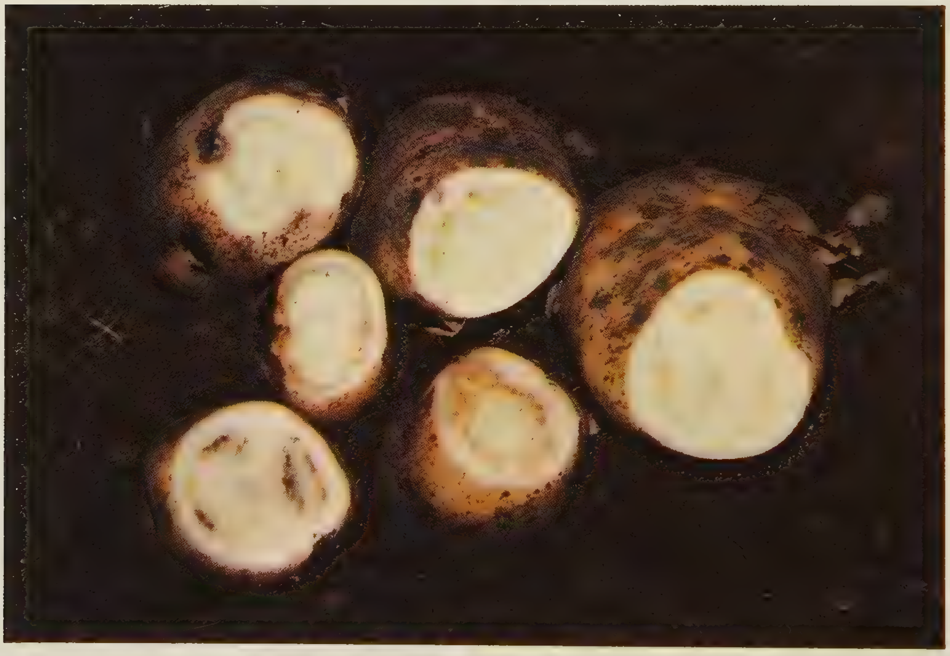
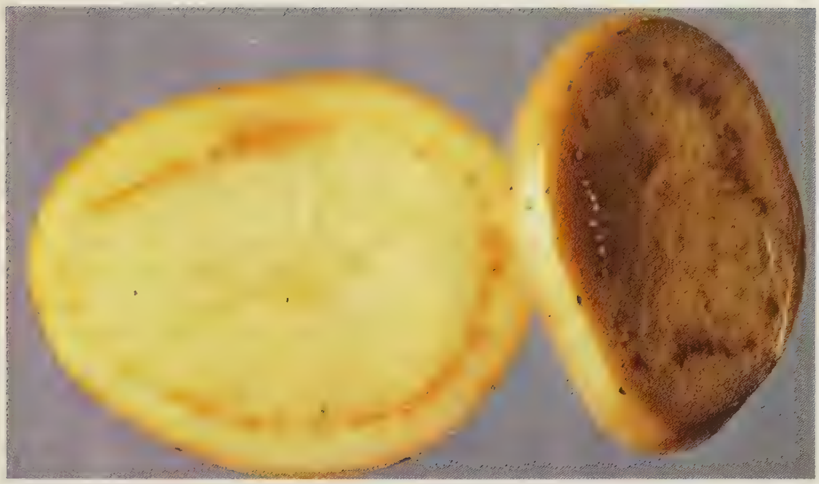


OCCURRENCE This bacterial disease is widespread in tropical, subtropical and warm temperate regions, and may occasionally be a problem in cooler latitudes (10). Several strains of the bacterium exist, adapted to different climatic conditions (6). Records include India, Pakistan (1), South Africa, Kenya (8), Ruanda, Egypt (5,9), Israel, Cyprus, Greece, Turkey, Portugal (7), Sweden, the USA (2), Central and South America (3,6) and Australia (4). In some countries (including the UK) brown rot is a notifiable disease, and infected potato shipments (5) are subject to statutory action at the port of arrival.

SYMPTOMS If an infected tuber is cut across at the heel end (5), the vascular tissues usually appear as a brown ring. After some minutes (or hours) creamy drops of bacterial exudate may ooze out; this is characteristic of brown rot (7). By contrast, in bacterial ring rot (q.v.) the exudate becomes apparent only when squeezed out by manual pressure. Another difference is that in tubers with severe brown rot infection, bacteria may exude through the eyes and stem-end attachment, causing soil particles to adhere. Bacteriological tests in the laboratory are necessary to confirm the diagnosis.



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BIOLOGY The brown rot organism can be transmitted in seed tubers (1), which may be symptomless (3). The bacteria persist in infected crop debris and also survive in the soil (4). Some strains infect certain weeds (4). The bacteria are water-borne, and disease development is favoured by high soil-moisture (9) and warm wet weather, the optimal temperature being about 25°C. Infection occurs via injuries in roots or stems, and through natural openings (stomata) in the leaves. Subsequently the bacteria multiply and invade the plant's vascular system, causing a wilt (9). Susceptible plants may die; alternatively, plants of healthy appearance may give rise to infected tubers (3).

CONTROL The use of clean seed is essential (1,3). In infested areas a long rotation is beneficial, and it should be noted that tomatoes, peppers, eggplants and tobacco are also subject to this disease (2). Infested debris should be destroyed, and it may be necessary to eradicate weeds (4). Soil disinfestation is impracticable (4), but improvements in soil drainage can be worthwhile (9). Quarantine measures are important in helping to prevent further spread of the brown rot organism (10). Potato cultivars differ in their susceptibility to brown rot, and work is in progress to breed resistant cultivars (10).

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See also:

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CHARCOAL ROT of potatoes caused by

Macrophomina phaseolina **(Tassi) Goid.**

Sclerotial state: *Sclerotium bataticola* Taubenh.

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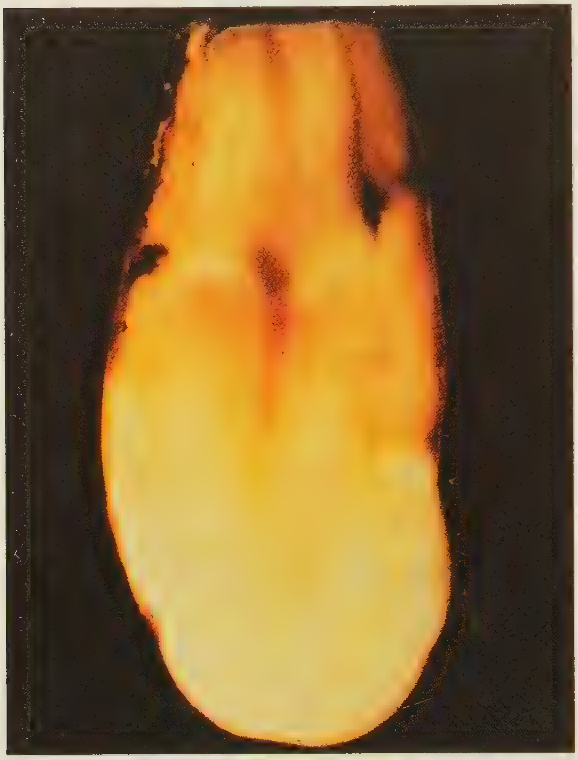

OCCURRENCE Charcoal rot is especially important in tropical and subtropical countries. It has caused serious losses in India (1,3,4) and the southern USA (5), and also in Mediterranean countries such as Cyprus, Egypt and Israel (2). The disease has been recorded only occasionally in temperate countries, for example France and Germany.

SYMPTOMS Black spots develop on the tuber surface, centred on the natural openings (lenticels) in the skin (2). Alternatively, or in addition, blackening occurs around the eyes and at the place where the tuber was attached to the plant (5). Lesions may also be associated with injuries (1). Affected flesh is grey, buff or brown, often with a black border between diseased and healthy tissue. The fungus may invade the entire tuber, sometimes accompanied by other organisms, resulting in a soft wet rot or a dry rot with cavities (4,5).

BIOLOGY The fungus persists in the soil as small (up to 1 mm) spherical resting bodies (sclerotia) which, under suitable conditions, give rise to mould strands. Vigorous potato plants are resistant to attack, but if weakened by drought (2) or waterlogging (5) they may be invaded via the stem, roots or tubers. Infection occurs only during hot weather, the optimal temperature for disease development being 30° to 35°C (1,3,5). Sometimes, in stem tissue, the fungus produces the asexual state (pycnidia giving rise to conidia), and the spores can survive for several weeks in the soil (1), initiating new infections if conditions remain favourable. Decay in tubers may be apparent at harvest time or may develop in store, where there may be further spread of the fungus by tuber moths (3). The disease can be carried to new areas in infected tubers.

CONTROL It may be necessary to treat planting material with a chemical or bacterial preparation (4). Since most potato cultivars are susceptible to charcoal rot, the usual strategy is to avoid disease by choosing early maturing cultivars and planting as early as possible so that the crop is ready for harvest before the onset of hot weather (4,5). Frequent irrigation serves to cool the soil (4) as well as to prevent water-stress in the plants (2); at the same time, good drainage is also important (5). Tubers should be harvested and handled with care to prevent injury (1). Storage at temperatures below 10°C provides complete control of post-harvest rotting by this fungus, but decay ensues if infected tubers are returned to ordinary temperatures (1,5). Work is in progress to breed potatoes with resistance to charcoal rot.

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4 THIRUMALACHAR M.J. & O'BRIEN M.J. (1977) Suppression of charcoal rot in potato with a bacterial antagonist. *Plant Disease Reporter* **61,** 543–546.

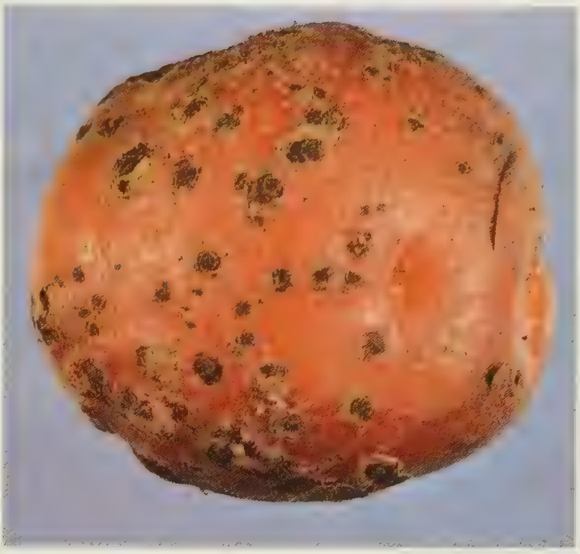
5 WATSON R.D. (1944) Charcoal rot of Irish potatoes. *Phytopathology* **34,** 433–435 illus.

COMMON SCAB of potatoes caused by
Streptomyces scabies **(Thaxt.) Waksman & Henrici**

OCCURRENCE Common scab is widespread but tends to be most important in countries where a high value is placed on external appearance and skin quality of potatoes; blemished tubers are likely to be down-graded. Scabbed seed tubers may be rejected for export, since many countries have strict regulations regarding the occurrence of scab on imported seed potatoes (4). Such regulations may be unjustified, in view of the fact that the organism is already present in most areas; furthermore, even severely scabbed seed tubers have been shown to produce normal yields. Investigations have been carried out in, for example, the UK (1), Switzerland (2), the Netherlands (4), India (5) and the USA (3). *S. scabies* is an actinomycete, a bacterium with some of the properties of a fungus. Related species (2) are responsible for other types of scab, for example netted scab (q.v.) and russet scab (q.v.).

SYMPTOMS Common scab lesions are dark brown, circular to star-shaped roughened areas which may be superficial or deep, according to the virulence of the organism and the susceptibility of the potato cultivar (4). Affected tissue is dry, corky and cracked, sometimes resembling the early stage of powdery scab (q.v.).

BIOLOGY The common scab organism is a common and persistent soil inhabitant, having been introduced to most areas on infected seed tubers (4). It survives either as spores or as slender strands within crop debris, and can be spread by water or by windblown soil. Developing potato tubers are infected via wounds or, more commonly, through natural openings (young lenticels recently formed from stomata) (1). The surrounding tissue reacts to invasion by producing layers of cork cells which give the lesions their characteristic scabby appearance (4). Infection (which is optimal at temperatures between 19° and 24°C) is facilitated by dry soil conditions during the period of tuber formation, probably because of the resulting paucity of antagonistic bacteria (1,4). Lesions are fully apparent at harvest time and do not progress in store, though deep scab lesions may predispose the tubers to invasion by secondary decay organisms.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CONTROL Potatoes should not be grown repeatedly on the same land, lest the soil become increasingly infested (4). Crop debris should be destroyed after harvest. Other cultural methods include the ploughing in of certain green manures. Also effective are the use of soil disinfectants and the application of growth-retardant chemicals as foliar sprays. The most practical control measure, however, is to provide adequate irrigation for several weeks from the time of tuber initiation, since wet soils inhibit infection by the common scab organism (1,4) (cf. powdery scab, q.v.). Potato cultivars differ greatly in their susceptibility, some showing high resistance (3,4).

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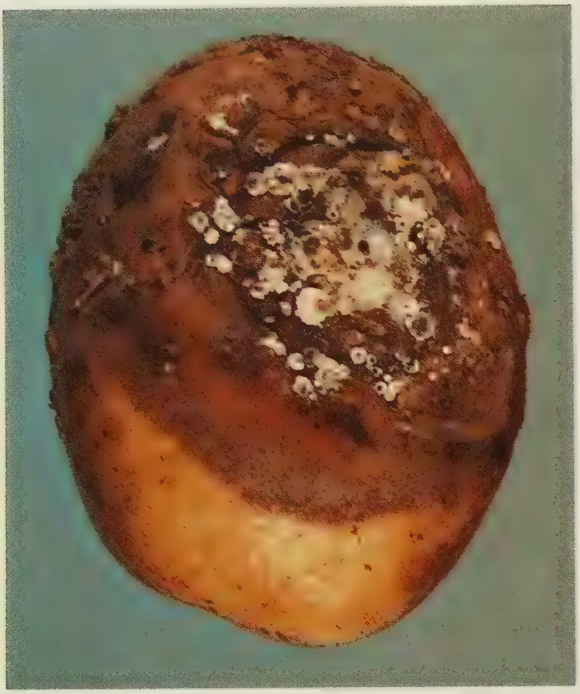
FUSARIUM ROT of potatoes caused by

***Gibberella* spp.**

Conidial state: *Fusarium* spp.

419



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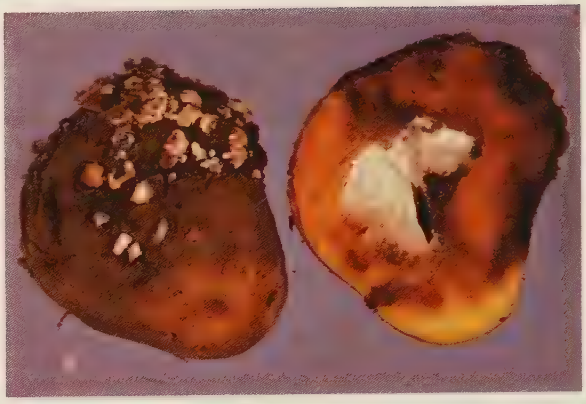
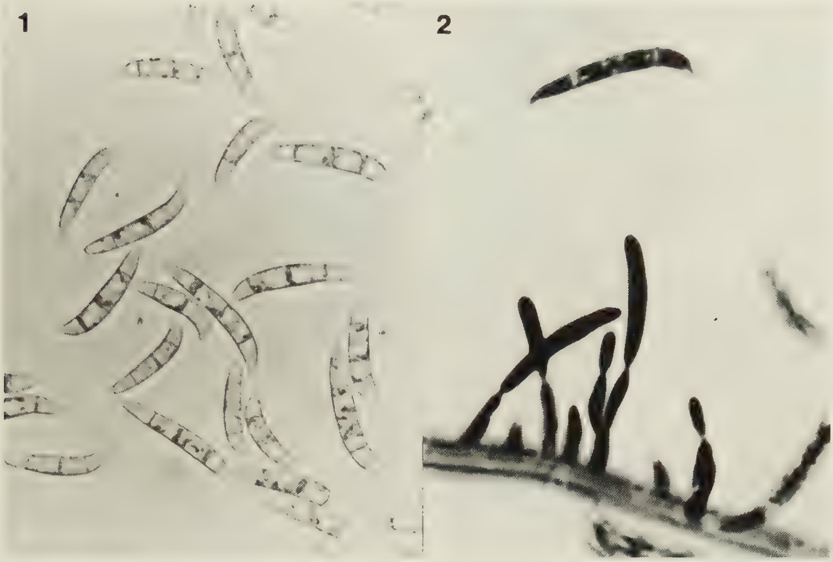


OCCURRENCE In any potato-growing area there may be several species of *Fusarium*, with one or two predominating (8). *F. solani* var. *coeruleum* (Sacc.) Booth is prevalent in Europe (2,7,9) and the northern USA (5), and has been responsible for heavy losses in store (4). Other important species are *F. sulphureum* Schlecht. (syn. *F. sambucinum* f.6), which is common in the USA (5), the UK (1,4) and Cyprus, for example, and *F. trichothecioides* Wollenw., found in Australia, western Canada and the USA, and several parts of Europe (8). Other records include *F. arthrosporioides* Sherb. in France (9), *F. avenaceum* (Corda ex Fr.) Sacc. in Finland (8), *F. equiseti* (Corda) Sacc. in India (6), *F. oxysporum* Schlecht. in Egypt and South Africa, and *F. sporotrichioides* Sherb. in Egypt and Northern Ireland (10). Numerous other species can cause rotting in potato tubers (8).

SYMPTOMS The nature of the rot depends both on the species involved and on the storage environment (8). There may be a single dark lesion or else several small lesions centred on natural openings (lenticels) and later coalescing (6). The margin of the affected area may be well-defined or quite diffuse. Frequently a dry mealy rot ensues and, indeed, dry rot is the common name for this disease. The skin tends to form concentric wrinkles as the tissue loses moisture, while the flesh develops cavities lined with a pale-coloured spore-bearing mould. In a humid atmosphere some species of *Fusarium* can cause a moist, firm rot (8) or even a soft rot (10). Soft-rotting may also occur after secondary infection by bacteria (4).

BIOLOGY All these fungi are soil inhabitants (9), often persisting as thick-walled resting spores (chlamydospores). Germination of these spores appears to be stimulated by exudates from the roots of potato plants (3); mould strands develop and produce asexual spores (conidia) which contaminate the developing tubers. Immature tubers, if harvested whilst the foliage is still green, are susceptible to infection (via wounds) and immediate decay (2). Mature tubers are resistant to decay, but only for a limited period, becoming susceptible after several weeks or months in store (2,5). If wounds sustained at harvest time are harbouring latent infection, then losses after a period of storage can be substantial (7). Optimal temperatures for decay differ according to species e.g. 20°C for *F. solani* var. *coeruleum* and 30°C for *F. avenaceum*. *F. sulphureum* and *F. trichothecioides* have two optimal temperatures for decay, viz. 12° and 25°C, no doubt a reflection of the variable susceptibility of the tuber at different temperatures (8). New infections can take place if, after storage, the crop suffers further mechanical damage during grading. Seed potatoes are often infected at this stage, or subsequently if cut into seed pieces (4). Whether infected or merely contaminated (1), seed tubers can transmit the fungus to new areas (9). They may rot without producing a plant, resulting in 'gappy' crops. Alternatively, the spores which (according to species) form on decaying seed tubers or on infected stem-bases subsequently contaminate the developing daughter tubers (1,9).



421 *Fusarium sulphureum* (from CMI Descr. No. 574, Booth 1978)
1 conidia × 500 2 conidia and conidiophores × 750

CONTROL Immature tubers are best protected by allowing a period of at least two weeks between 'burning off' the foliage (page 297) and harvesting the crop; during this time they become resistant (2). Particular care should be taken to minimise damage during harvesting and handling. Traditional curing (page 298) of mature tubers, while beneficial in controlling other potato diseases, is not always effective against fusarium rot, which is best controlled by the use of a fungicide dust, dip or spray (4). It is essential to apply the chemical before the fungus becomes established in wounds; hence treatment should be carried out directly after harvest or, in particular, immediately following grading out of store (4). Potato cultivars differ in susceptibility to the disease, and work is in progress to breed cultivars with resistance to several species (5).

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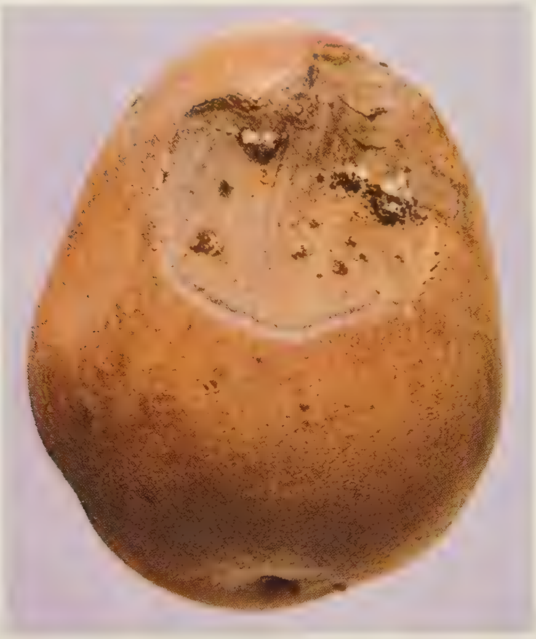
1 ADAMS M.J. & LAPWOOD D.H. (1983) Transmission of *Fusarium solani* var. *coeruleum* and *F. sulphureum* from seed potatoes to progeny tubers in the field. *Annals of Applied Biology* **103,** 411–417.
2 BOYD A.E.W. (1967) The effects of the length of the growth period and of nutrition upon potato tuber susceptibility to dry rot (*Fusarium caeruleum*). *Annals of Applied Biology* **60,** 231–240.
3 GÜLSOY H.E. (1979) Behaviour of the chlamydospores of *Fusarium solani* var. *coeruleum* (Sacc.) Booth in soil. *Journal of Turkish Phytopathology* **8**(2/3), 81–96.
4 HIDE G.A. & CAYLEY G.R. (1985) Effects of delaying fungicide treatment of wounded potatoes on the incidence of *Fusarium* dry rot in store. *Annals of Applied Biology* **107,** 429–438.
5 LEACH S.S. & WEBB R.E. (1981) Resistance of selected potato cultivars and clones to *Fusarium* dry rot. *Phytopathology* **71,** 623–629.
6 RAI R.P. (1979) *Fusarium equiseti* (Corda) Sacc. causing dry rot of potato tubers – a new report. *Current Science* **48,** 1043–1045 illus.
7 SCHOENE K. (1967) Untersuchungen zur Anfälligkeit von Kartoffelknollen gegenüber *Fusarium coeruleum* (Lib.) Sacc., dem Erreger der Weissfäule. *Phytopathologische Zeitschrift* **60,** 201–236 illus.
8 SEPPÄNEN E. (1981) Fusariums of the potato in Finland. 2. On the growth optima of *Fusarium* species in the tubers of cv Bintje. *Annales Agriculturae Fenniae* **20,** 161–176 illus.
9 TIVOLI B., JOUAN B. & LEMARCHAND E. (1986) Étude de la transmission à la descendance de différentes espèces ou variétés de *Fusarium* responsables de la pourriture sèche des tubercules de pomme de terre: rôle du tubercule de semence, du sol et de la plante. *Potato Research* **29,** 141–162.
10 UPSTONE M.E. (1970) A potato tuber rot caused by *Fusarium sporotrichioides* Sherb. *Plant Pathology* **19,** 150.

See also:

THERON D.J. & HOLZ G. (1990) Effect of temperature on dry rot development of potato tubers inoculated with different *Fusarium* spp. *Potato Research* **33,** 109–117.

GANGRENE of potatoes caused by
Phoma exigua **Desm. var.** ***exigua***
Phoma exigua **Desm. var.** ***foveata***

422



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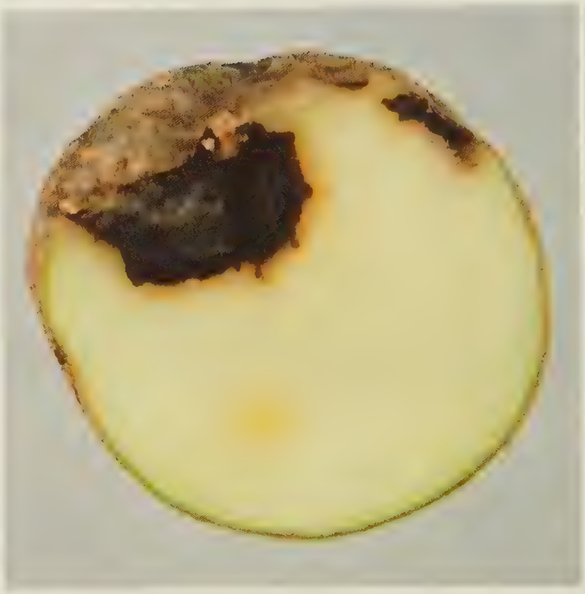
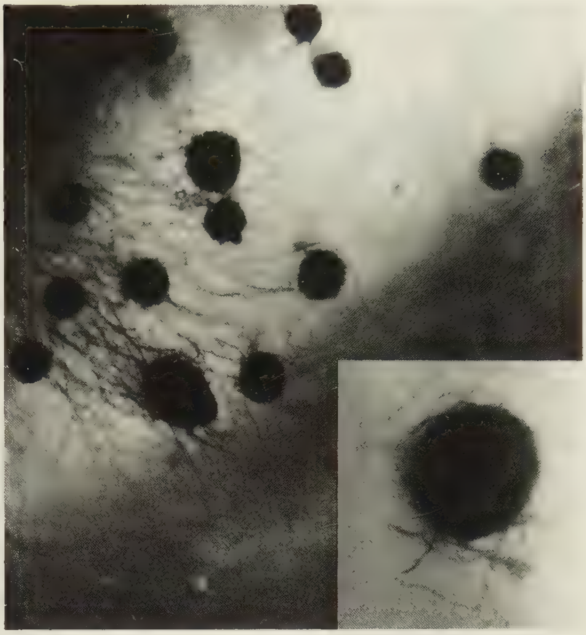


OCCURRENCE Gangrene has caused significant storage losses in the UK (1,3,4,5,8,9). The disease also occurs in other European countries such as Sweden (2) and Germany (7), and in the USA, Canada and Australia (10). Although more usually associated with stored mature potatoes, the disease is occasionally seen in immature tubers and has been recorded on Egyptian 'new' potatoes imported into the UK (6).

SYMPTOMS Small (up to 1 mm) circular 'target spots' may develop, centred on lenticels (6). Such lesions are characterised by a dark brown centre and a buff-coloured halo bordered by a fine dark margin. Alternatively, much larger dark sunken lesions develop (typically at wound sites), to which the common name 'devil's thumb-print' has been given. Minute black bodies (pycnidia) may be visible on the surface and, in a humid atmosphere, a white mould develops (7). The restricted lesions often conceal substantial internal decay. Rotted tissue is dark, sometimes with a purplish hue, and is sharply delimited from healthy tissue (6). Cavities are formed if the flesh dries out during storage, and pycnidia may develop within. *P. exigua* var. *exigua* is synonymous with *Ascochyta phaseolorum* (page 107). It causes less extensive tuber rots than *P. exigua* var. *foveata*, which is chiefly confined to potato and is distinguished in culture by its ability to produce pigment (8,9).

BIOLOGY The fungi can be transmitted on or in seed tubers (6), and this is especially true of *P. exigua* var. *foveata*. Severely infected seed tubers may undergo a watery decay in the ground, with copious production of asexual spores (conidia discharged from pycnidia) (8). Spores are dispersed by rain and irrigation water, and can cause infection of developing daughter tubers via lenticels (6). Slightly infected seed tubers give rise to apparently healthy plants. However, shortly before harvest, when the haulm is moribund, stem lesions develop, with numerous pycnidia (9). Spore discharge is favoured by wet conditions, and spores are dispersed in wind-blown rain drops. Dry spores may also be wind-dispersed, and *P. exigua* var. *exigua* appears to be distributed more widely by this means (9). Harvested tubers are often severely contaminated (2), or may be carrying latent infection when placed in store. The most important factors influencing post-harvest rotting are the incidence (and nature) of mechanical damage and the initial storage regime (1,10). Injuries to the tuber skin open the way for invasion, and a high incidence of gangrene can be related to wounds sustained during mechanical harvesting and grading (4). Temperature and humidity determine the rate of wound-healing as well as the rate of growth of the fungus, and at temperatures between 0° and 5°C the formation of a protective corky barrier is inhibited (1,7,10). Gangrene is therefore especially prevalent after potatoes have been lifted in cold weather and kept under cool conditions. Susceptibility to decay increases with length of time in store (2).

CONTROL Clean seed can be obtained by means of stem-cuttings, but there is always a possibility of the stock becoming contaminated by spore-laden dust from potato stores. With regard to disease control in the field, the application of foliar fungicide sprays has had variable results, and other measures are preferred. The crop should be harvested as soon as is feasible. In countries such as the UK, an early lifting date makes it less likely that the tubers will be subjected to frosty weather and to the increased mechanical damage which is often associated with harvesting under such conditions. It is advisable to lift the crop fairly promptly after 'burning off' (page 297), so that the tubers escape contamination by spores which eventually form in the weakened haulm (3,4,9). Alternatively the haulm can be removed from the field before harvest begins. Injury to the tubers can be minimised by correct adjustment and maintenance of machinery used for harvesting and grading (4). Prompt application of a post-harvest fungicide is beneficial; the tubers can be dipped or sprayed (3). It is highly desirable to provide suitable conditions for efficient curing (page 297), wound-healing being accomplished in about 2 weeks at moderate temperatures (above 10°C) and high humidity (1,7,10). Subsequently the tubers can be stored under cool conditions. If it is not possible to cure potatoes properly, an alternative strategy is fumigation (5). This can be very effective provided that a sufficiently forceful air circulation system is available and the tubers are largely free of soil and debris. The technique can be adapted to boxed or bulk-stored potatoes (whether seed or ware), and the fumigant should be applied within a few days of harvest (5).



424 *Phoma exigua* var. *foveata* (from *Trans. Br. mycol. Soc.* **71**, 319–320, Lapwood, Harris & Hide 1978) Pycnidia forming in water from rotting tuber tissue (insert showing ostiole)

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1 ADAMS M.J. & GRIFFITH R.L. (1983) The effect of temperature before or after inoculation with *Phoma exigua* var. *foveata* on the development of gangrene in potato tubers. *Plant Pathology* **32,** 325–333.

2 BÅNG U. (1986) Effects of planting potato tubers attacked by *Phoma exigua* var. *foveata* on yield and contamination of progeny tubers. *Potato Research* **29,** 321–331.

3 CARNEGIE S.F., HIDE G.A., RUTHVEN A.D., LINDSAY D.A. & ADAMS M.J. (1988) Control of potato gangrene by thiabendazole in relation to time of harvest after haulm destruction and site of production. *Annals of Applied Biology* **113,** 471–481.

4 COPELAND R.B. (1982) The influence of potato harvesting and grading machinery on contact spread of *Phoma exigua* var. *foveata* on tubers. *Annals of Applied Biology* **101,** 465–472.

5 HAMILTON G.A. (1985) Fumigation of potatoes with 2-aminobutane for control of gangrene and skin spot. *EPPO Bulletin* **15,** 25–28.

6 HIDE G.A. (1986) *Phoma* and *Fusarium* rots on imported "new" potatoes. *Plant Pathology* **35,** 126–127 illus.

7 KRANZ J. (1958) Untersuchungen über die Phoma-Fäule der Kartoffelknolle unter besonderer Berücksichtigung des Wirt-Parasit-Verhältnisses. *Phytopathologische Zeitschrift* **33,** 153–196 illus.

8 LAPWOOD D.H., HARRIS R.I. & HIDE G.A. (1978) Isolation of *Phoma exigua* var. *foveata* from, and pycnidial formation on, brown watery rots of planted seed potato tubers. *Transactions of the British Mycological Society* **71,** 319–320 illus.

9 LOGAN C. (1976) The spread of *Phoma exigua* within the potato crop. *Annals of Applied Biology* **82,** 169–174.

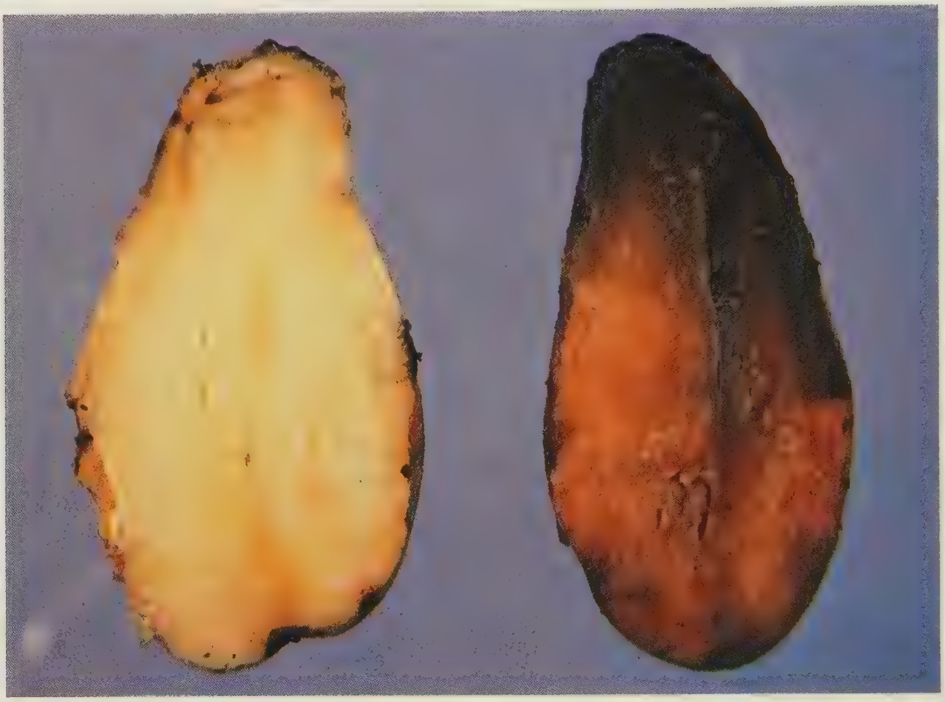
10 WALKER R.R. & WADE G.C. (1978) Resistance of potato tubers (*Solanum tuberosum*) to *Phoma exigua* var. *exigua* and *Phoma exigua* var. *foveata. Australian Journal of Botany* **26,** 239–251 illus.

See also:

BAIN R.A., LENNARD J.H. & WASTIE R.L. (1988) The influence of cultivar and mechanical damage on the relationship between inoculum concentration of *Phoma exigua* var. *foveata* on potato tubers and gangrene development. *Plant Pathology* **37,** 265–270.

PINK ROT of potatoes caused by *Phytophthora erythroseptica* Pethybr.

425

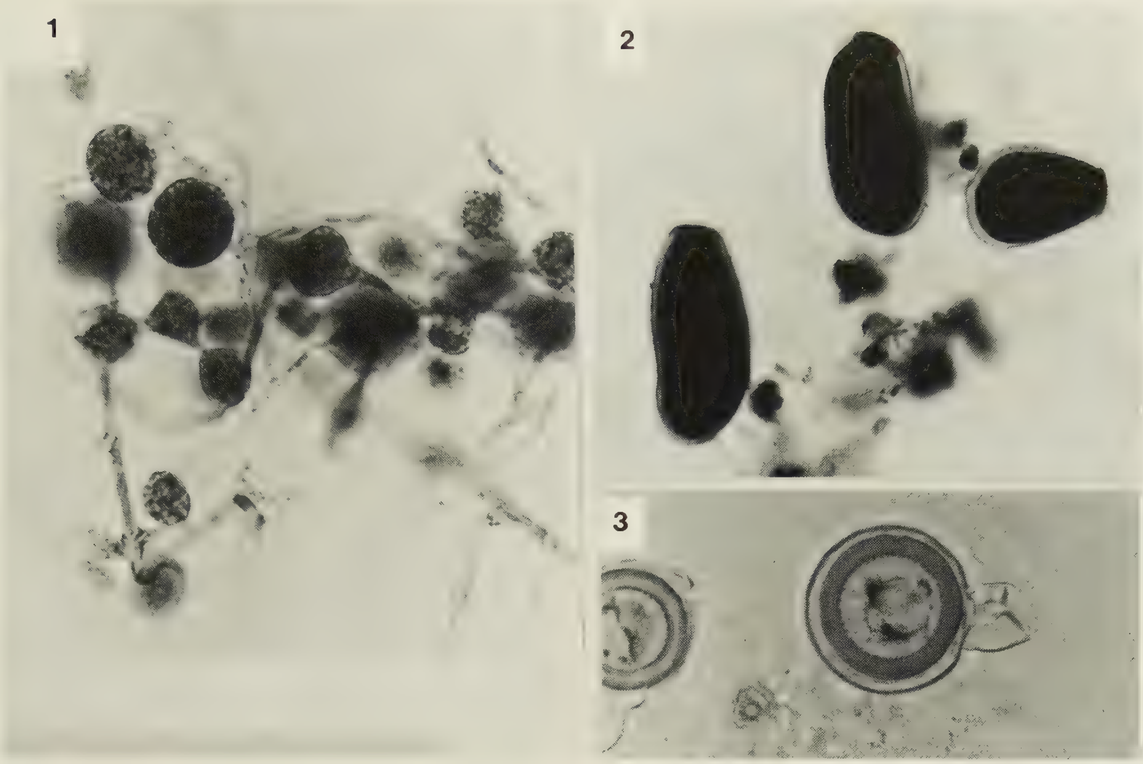


OCCURRENCE The disease occurs worldwide, but outbreaks tend to be sporadic and localised (3). *P. erythroseptica* is usually responsible, and records include Ireland (2), the UK (2,6,7), Germany (5), Switzerland (9), Egypt (1), Peru (10), the USA (3,8) and Canada (4) (CMI Map 83). Other species which can cause pink rot include *P. cryptogea* Pethybr. & Lafferty (8) and *P. drechsleri* Tucker (3) in the USA, and *P. megasperma* Drechsler in the UK (2).

SYMPTOMS Infected tubers are rubbery in texture but not soft (4). Decay usually begins at the heel end, and the lesion is often bordered by a black margin (3). The natural openings (eyes and lenticels) are discoloured (1) and may exude a sticky fluid to which soil particles adhere (4). If a partially rotted tuber is cut in half and exposed to air for 15 to 20 minutes, the healthy tissue retains its normal colour but infected tissue turns pink, later darkening to a purplish black (1,3). Such a colour change may also be associated with bacterial soft rot (q.v.) and rubbery rot (q.v.); in pink rot, however, there is often, in addition, a characteristically pungent odour (3,9). If the flesh is compressed, a clear fluid is expelled and the tissue does not regain its former shape (3).

BIOLOGY These fungi persist in soil and debris, usually in the sexual state (thick-walled oospores) (1,9). Transmission in infected seed tubers has always been considered unlikely, since rapid rotting after harvest precludes their use as planting material (6). However, sound seed tubers may be superficially contaminated with oospores, which are thus transferred to new areas (7). In warm wet conditions (10) the fungi produce the asexual state (sporangia and sometimes zoospores), and these spores are dispersed by rain or irrigation water (cf. blight, q.v., caused by *P. infestans*, in which sporangia are dispersed by air). Roots, stem-bases and stolons may become infected, and hence the progeny tubers (3, 7). In waterlogged soils direct infection can occur via eyes and lenticels (2) and tubers rot in the field. The only above-ground symptom is a late-season wilt (8, 9,10). Post-harvest rotting can be very severe (2); in a warm moist storage atmosphere decay spreads rapidly into neighbouring tubers (3) and secondary bacterial soft rot (q.v.) often follows.

CONTROL A long rotation is desirable, with non-susceptible crops, and prompt removal of debris is also important in reducing the level of soil infestation (2). Fumigation of soil has been recommended, together with the use of a systemic fungicide applied in the furrow at the time of planting. It may be necessary to improve drainage or adjust the irrigation system so that water does not accumulate (3, 4). If it is feasible to do so, wilted plants should be promptly removed and destroyed (9). Early lifting



426 *Phytophthora erythroseptica* (from CMI Descr. No. 593, Stamps 1978) 1 hyphal swellings in water 2 sporangia 3 oogonium with amphigynous antheridium and oospore × 500

ensures a minimum level of infection in harvested tubers (2,9), but slightly infected tubers escape detection and later rot in store (6). If it is suspected that pink rot may be a problem, extra care should be taken to provide thorough ventilation in the store (2), so that infected tubers will become mummified rather than undergoing a wet rot. Potato cultivars differ in susceptibility to pink rot, but none is immune (2,6).

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1 ABO-EL-DAHAB M.K. & EL-GOORANI M.A. (1969) The pink rot disease of potato tubers in U.A.R. (Egypt). *Journal of Phytopathology of the U.A.R.* **1,** 53–58 illus.

2 CAIRNS H. & MUSKETT A.E. (1933) Pink rot of the potato. *Annals of Applied Biology* **20,** 381–403 illus.

3 GOSS R.W. (1949) Pink rot of potatoes caused by *Phytophthora erythroseptica* Pethyb. *Research Bulletin of Nebraska Agricultural Experiment Station* No. 160, 27 pp illus.

4 JONES W. (1954) Pink rot of potato tubers on Vancouver Island. *Canadian Journal of Agricultural Science* **34,** 504–506 illus.

5 KLEINHEMPEL D., PETT B. & EFFMERT M. (1981) Auftreten der Rotfäule (*Phytophthora erythroseptica* Pethybr.). *Archiv für Phytopathologie und Pflanzenschutz* **17,** 351–354 illus.

6 LENNARD J.H. (1980) Factors influencing the development of potato pink rot (*Phytophthora erythroseptica*). *Plant Pathology* **29,** 80–86.

7 LONSDALE D., CUNLIFFE C. & EPTON H.A.S. (1980) Possible routes of entry of *Phytophthora erythroseptica* Pethyb. and its growth within potato plants. *Phytopathologische Zeitschrift* **97,** 109–117 illus.

8 ROWE R.C. & SCHMITTHENNER A.F. (1977) Potato pink rot in Ohio caused by *Phytophthora erythroseptica* and *P. cryptogea*. *Plant Disease Reporter* **61,** 807–810 illus.

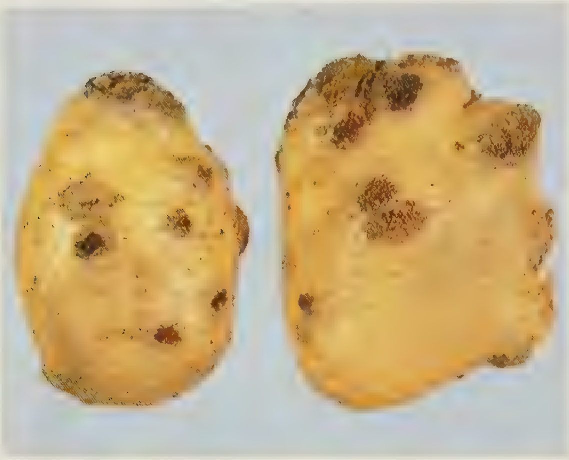
9 SIJAK A. & GINDRAT D. (1969) Quelques cas de "pourriture rose" (pink rot) de la pomme de terre en Suisse romande. *Revue Suisse d'Agriculture* **1,** 151–153 illus.

10 VARGAS L.A. & NIELSEN L.W. (1972) *Phytophthora erythroseptica* in Peru: its identification and pathogenesis. *American Potato Journal* **49,** 309–320.

POWDERY SCAB of potatoes caused by

Spongospora subterranea **(Wallr.) Lagerh. f.sp. *subterranea* Tomlinson**

427



OCCURRENCE The fungus has a wide distribution, but the disease is prevalent only in cool wet conditions (2,3). Powdery scab is important in several European countries, especially those with a seed export trade such as the Netherlands (3) and the UK (5). Other records include Austria and Switzerland (2). In most parts of the USA powdery scab is of minor importance. In India it occurs in the hills but not in the warmer lowlands (1). In Israel and Australia disease incidence has increased since susceptible cultivars have been grown in winter with the help of irrigation (4). As well as disfiguring the tubers, the fungus may also transmit a virus which causes 'mop-top' symptoms in the growing crop (2).

SYMPTOMS The first signs on the tuber are raised purplish-brown spots about 1 mm in diameter (3). As the lesions enlarge, the skin eventually ruptures, exposing masses of dusty brown 'spore-balls' in characteristic pustules (2). In severe attacks the tuber may be deformed by cankers (5).

BIOLOGY The 'spore-balls' (cystosori) (**348**) are very persistent, and can survive in soil for at least 10 years, even in the absence of potato crops (2) (CMI Descr. 477). Under cool, moist conditions they produce swimming spores (zoospores) which attack the root hairs of susceptible plants, in which they produce irregular multinucleate bodies (plasmodia giving rise to zoosporangia). More zoospores are released; they fuse in pairs or threes and are capable of infecting young potato tubers through injuries or natural openings (lenticels); this results in pustule formation. The eyes are especially susceptible to infection, and invaded tissue may be stimulated into abnormal growth, with the formation of large tumours or cankers (5). Scabbed tubers are sometimes predisposed to invasion by the late blight fungus or other organisms. After harvest, existing scabs and cankers may develop further, but adjacent healthy tubers do not become infected.

CONTROL Crop rotation has proved unsatisfactory owing to the longevity of the organism (2). Improvements in drainage can be worthwhile. It is not usually feasible to treat infested soil, although some chemicals are partially effective (1). Seed tubers, whether infected or merely contaminated, should be soaked in a fungicide and planted in clean land. Since potatoes are especially vulnerable during the 3 to 4 weeks following tuber initiation (4) it can be advantageous to withhold irrigation during this period (cf. the measure against common scab, q.v.). Different cultivars vary from highly susceptible to highly resistant, and breeding work continues (5).

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1 BHATTACHARYYA S.K. & RAJ S. (1986) Field screening of different chemicals against powdery scab of potatoes. *Pesticides* **20**(2), 23–26.
2 DIRIWÄCHTER G. & GINDRAT D. (1982) Contribution à l'étude de la gale poudreuse de la pomme de terre. *Revue Suisse d'Agriculture* **14,** 265–269 illus.
3 KOLE A.P. (1954) A contribution to the knowledge of *Spongospora subterranea* (Wallr.) Lagerh., the cause of powdery scab of potatoes. *Tijdschrift over Plantenziekten* **60,** 1–65 illus.
4 TAYLOR P.A., FLETT S.P., BOER R.F. de & MARSHALL D. (1986) Effects of irrigation regimes on powdery scab disease and yield of potatoes. *Australian Journal of Experimental Agriculture* **26,** 745–750.
5 WASTIE R.L., CALIGARI P.D.S. & WALE S.J. (1988) Assessing the resistance of potatoes to powdery scab (*Spongospora subterranea* (Wallr.) Lagerh.). *Potato Research* **31,** 167–171.

See also:

NACHMIAS A. & KRIKUN J. (1988) Etiology and control of powdery scab of potato in a semi-arid region of Israel. *Phytoparasitica* **16,** 33–38.

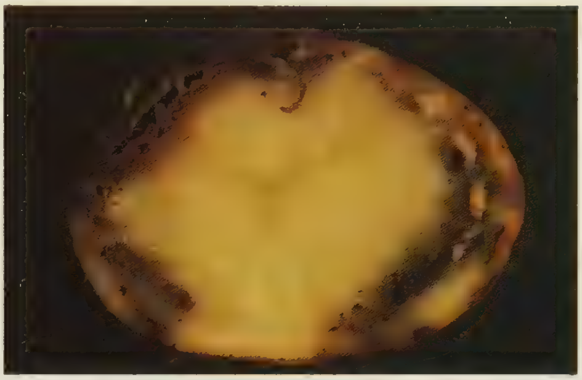
RING ROT of potatoes caused by

Clavibacter michiganensis **ssp. *sepedonicus*** (Spiek. & Kotth.) Davis et al.
(syn. *Corynebacterium sepedonicum* (Spiek. & Kotth.) Skapt. & Burkh.)

OCCURRENCE Ring rot is one of the most serious diseases of potatoes and can cause very heavy losses (1). The bacterium is generally confined to temperate regions (CMI Map 20), occurring in parts of the USA and Canada (1,4), and some European countries, for example Norway (5) and Denmark (2). It has been successfully eradicated from Germany and is not present in the UK. Strict quarantine regulations are in force in many countries (3).

SYMPTOMS Tubers should be examined by cutting them across near the heel end. In slightly infected tubers the vascular ring is barely discoloured. In more advanced infections the ring is brownish and the rotted vascular tissue has a creamy or cheesy consistency, especially noticeable if the tuber is gently squeezed. This is in contrast to brown rot (q.v.), in which a creamy bacterial exudate gradually oozes from the vascular strands. Specialised laboratory techniques are available for confirmation of identity (1,2). In common with brown rot, ring rot is an odourless decay, unless secondary bacteria cause further breakdown of the flesh. Severely rotted tubers exhibit surface cracks, together with dark blotches immediately beneath the skin.

BIOLOGY Bacteria persist in dried debris which is often present in potato sacks or on packhouse machinery (4). Contrary to earlier belief, the ring rot organism is not inactivated by freezing weather (4). The most important source of infection is contaminated or infected seed tubers (3,5), and further contamination can occur if the seed is cut into pieces in preparation for planting (4). Developing plants become infected throughout (systemic infection), the bacteria having been transported in the vascular tissues. Sucking insects can transmit bacteria to healthy plants. Infected leaves become rolled at the edges and stems may collapse. Some tubers appear normal at harvest time but carry latent infection (3,5).


428

CONTROL Co-ordinated efforts are required for the control of ring rot. Successful eradication in some European countries has been attributed mainly to the use of whole seed tubers. On the other hand, American and Canadian practices have enhanced dissemination of the ring rot organism (1). North American potato cultivars tend to have large tubers, and economy dictates that they be cut when used as seed; furthermore, a typical planting machine uses spikes to place individual seed pieces into the furrows. In all countries it is important to carry out regular disinfection of machinery and packhouse, together with other hygienic practices such as deep ploughing of crop debris (4). A strict seed certification scheme is vital. The sophisticated techniques devised for multiplying virus-free stocks (by means of meristem tip cultures) are of value in the control of ring rot (5). There are some resistant potato cultivars but these can be symptomless carriers and thus a constant threat to susceptible cultivars (5).

---

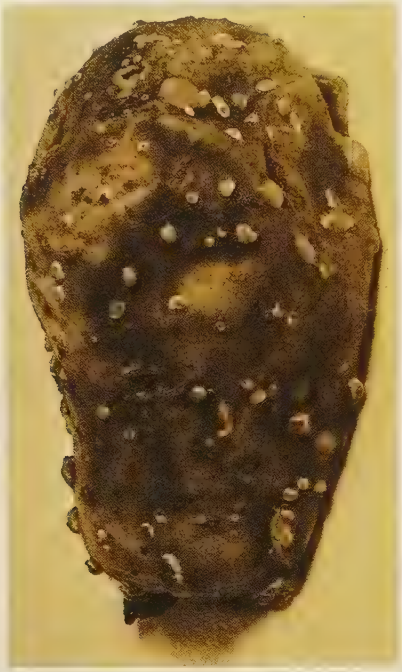
1 BOER S.H. de, HAAN T.-L. DE & MAWHINNEY J. (1989) Predictive value of post harvest serological tests for bacterial ring rot of potato. *Canadian Journal of Plant Pathology* **11,** 317–321.
2 DINESEN I.G. (1984) The extraction and diagnosis of *Corynebacterium sepedonicum* from diseased potato tubers. *EPPO Bulletin* **14,** 147–152.
3 MAZZUCCHI U., BAZZI C. & McKENZIE A.R. (1982) Transmission of *Corynebacterium sepedonicum* from latently infected seed potatoes in the Po valley. *Phytopathologia Mediterranea* **21**(2/3), 116–117.
4 NELSON G.A. (1984) Survival of *Corynebacterium sepedonicum* in potato stems and on surfaces held at freezing and above-freezing temperatures. *American Potato Journal* **62,** 23–28.
5 SLETTEN A. (1985) The effect of *Corynebacterium sepedonicum* on symptoms and yield of four potato cultivars. *Potato Research* **28,** 27–33.

See also:

GUDMESTAD N.C., SLACK S.A., MANZER F.E., BOER S.H. de, NELSON G.A., GUTBROT O., PRESTON D. & EASTON G.D. (1987) Proceedings of the Symposium on the eradication of bacterial ring rot (Colorado, 1986). *American Potato Journal* **64,** 663–701.

RUBBERY ROT of potatoes caused by
Geotrichum candidum **Link**

429



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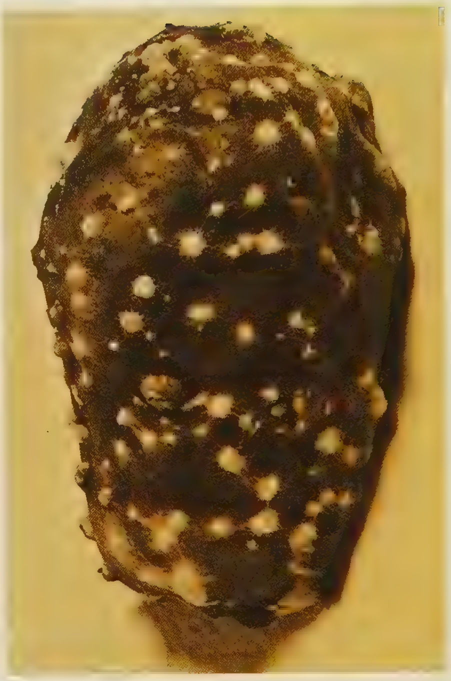

OCCURRENCE This disease has been recorded in several important growing areas in the UK and its incidence seems to be increasing (3,4). It has also been reported on Dutch seed potatoes imported into Italy (5).

SYMPTOMS Lesions appear as irregular brown patches with well-defined black margins. The entire tuber takes on a rubbery texture, and later releases a brownish liquid which has a distinct fishy odour (3). In a humid atmosphere greyish-white mould pustules develop at the natural openings (lenticels) or at breaks in the skin, and characteristic spores are produced (page 288) (2). If infected tubers are cut open, the flesh turns a greyish-pink colour after 3 to 4 hours (3). Rubbery rot is frequently followed by bacterial soft rot (q.v.).

BIOLOGY The fungus is a common soil inhabitant, and infection of potato tubers is favoured by warm wet conditions (3). Immature tubers are particularly susceptible if subjected to excessive irrigation shortly before harvest (3). Mature tubers are infected only via wounds (4). Symptoms may be apparent at harvest time, or may develop in store. The optimal growth temperature for the fungus is approximately 25°C (2) but it can also make progress at 10°C.

CONTROL Improved drainage may be necessary in order to prevent waterlogging of the soil (4). Heavy irrigation before harvest should be avoided, especially during very warm weather (3). A harvested crop known to be infected should be very thoroughly ventilated early in the storage period, in order to dry out infected tubers before they can develop into 'pockets' of soft rot (4).

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1 EL-TOBSHY Z.M. & SINCLAIR J.B. (1965) *Geotrichum candidum*: plant and animal isolates pathogenic to certain plant tissues. *Phytopathology* **55,** 1210–1212.

2 HOOG G.S. de, SMITH M.Th. & GUÉHO E. (1986) A revision of the genus *Geotrichum* and its teleomorphs. *Studies in Mycology* No. 29, 131 pp illus.

3 HUMPHREYS-JONES D.R. (1969) Rubbery rot (*Oospora lactis* (Fres.) Sacc.) of potatoes. *Plant Pathology* **18,** 186–187 illus.

4 LOGAN C. (1983) Potatoes. In *Post-harvest pathology of fruits and vegetables* (Ed. C. Dennis), pp. 179–217. London: Academic Press.

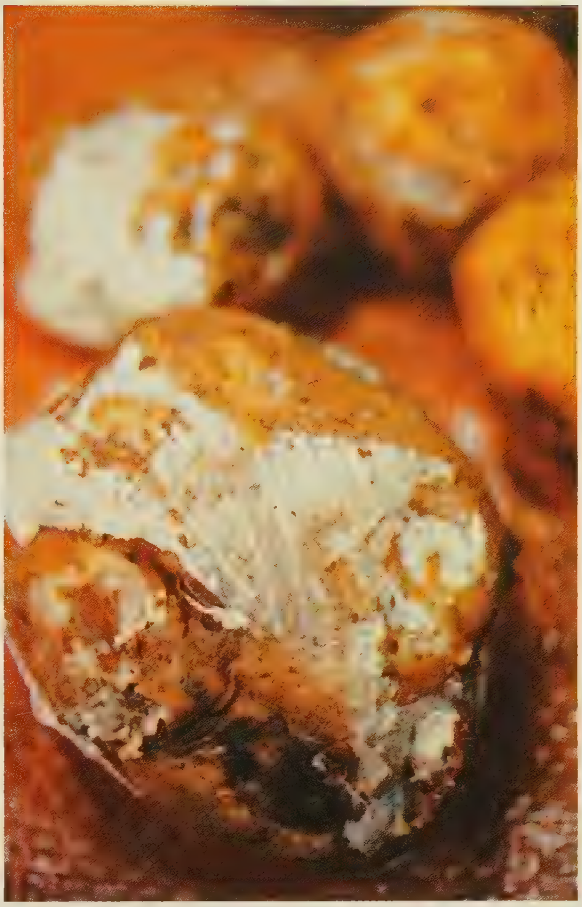
5 TOSI L. & ZAZZERINI A. (1986) Un marciume della patata causato da *Oospora lactis* (Fres.) Sacc. *Rivista di Patologia Vegetale* **22,** 126–129.

SCLEROTIUM ROT of potatoes caused by
Corticium rolfsii Curzi
Sclerotial state: *Sclerotium rolfsii* Sacc.

OCCURRENCE This disease is important in tropical and subtropical countries such as India (1) and the southern USA (2,5). It may also occur during spring and summer in warm temperate countries, for example Israel (3) and Italy (4).

SYMPTOMS Small sunken spots develop, usually centred on the natural openings (lenticels). Affected tissue becomes yellowish-brown and, as the lesions enlarge, they may support a silky white fan-shaped mould growth (4,5). In advanced stages of rotting the tuber tends to become spongy, with internal cavities containing small (1–3 mm) spherical white to brown resting bodies (sclerotia) (2,4). Alternatively a soft wet rot ensues, with leakage of fluid (2,5).

BIOLOGY Sclerotia persist in the soil and can be carried to new areas by irrigation water or farm implements (1). Under favourable conditions the sclerotia produce mould strands capable of attacking plants of any age. During warm wet weather infection usually occurs at the soil-surface, causing a stem-rot and wilt (1,2,4). More sclerotia are formed. The sexual state (basidia giving rise to basidiospores) occasionally develops on infected stem tissue. In dry conditions the fungus is more active below ground, and tends to attack the tubers directly (5). Severe losses may occur in the field. If tubers with incipient infection are harvested and stored in a warm environment, decay spreads rapidly into adjacent healthy tubers (4).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CONTROL Crop rotation is of limited value as a control measure, owing to the longevity of sclerotia and the susceptibility of most crop plants (1). It may be feasible to use soil treatments with the aim of killing the sclerotia before the crop is planted (1,3). Techniques (best used in combination) include the use of chemicals (1,3), solar heating of the soil (3) and the addition of an antagonistic fungus (biological control) (3). Seed pieces should be dipped in a fungicide (1). Planting date is important, since crops which can be harvested before the onset of hot weather generally escape the disease. During the growing season any diseased plants should be carefully removed and destroyed (4). If tuber infection is suspected, post-harvest decay can be temporarily arrested by refrigerated storage.

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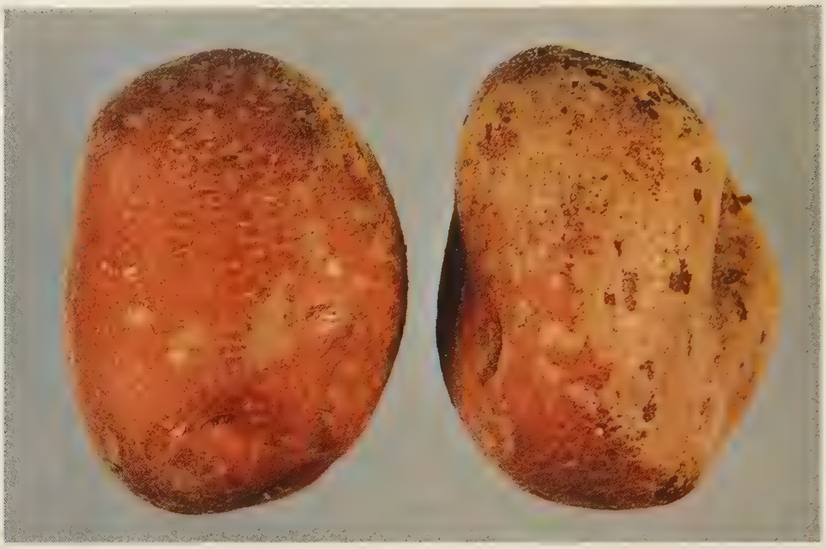
SILVER SCURF of potatoes caused by
Helminthosporium solani **Dur. & Mont.**

432



OCCURRENCE The fungus, which causes a skin blemish, probably occurs in all potato-growing countries (CMI Map 233), including the USA (1,2, 9), Canada (8), the UK (3,5,7), the Netherlands (6), France (4) and Israel (10). Silver scurf has assumed importance in recent years because it is especially obvious on tubers which have been washed for display in 'pre-packs' (3).

SYMPTOMS Brown patches develop on the tuber, sometimes confined to the heel end but often more generally distributed (1,9,10). Symptoms may not be apparent until the tuber is washed, when the lesions take on a silvery sheen (6). They are especially noticeable on coloured cultivars (9). In a humid atmosphere dark spores (conidia) are produced (1,2, 6,9), giving a sooty appearance, and this distinguishes silver scurf from black dot (q.v.); the two diseases may also occur together (3). Tubers with silver scurf become shrivelled owing to increased moisture loss (5,6,9).

BIOLOGY The fungus does not persist in soil, and its survival in crop debris is of little significance (8). The main source of infection is the seed tuber (5,7,8, 10). Crops produced from seed with severe scurf tend to be less affected than those produced from slightly infected seed (4,5,6). A possible explanation is that lesions on severely diseased seed may have lost the ability to produce many spores; on the other hand, abundant spores are produced over a longer period as small lesions extend over the surface of the mother tuber during plant growth and tuber production (7). Regardless of the severity of seed infection, the growth of the crop and its yield are practically unaffected (7). Infection of daughter tubers tends to occur only after the crop has already matured, penetration being via lenticels (1,9) or directly through the skin (by means of appressoria) (2). Even a low incidence of infection at harvest can nevertheless be a problem if the potatoes are to be stored (3,5); infected skin tissue is especially prone to moisture loss (5), and pieces may subsequently slough off (1,3). If infected potatoes are stored in a humid atmosphere at temperatures above 5°C, the lesions enlarge and there is copious spore production (3,5). New infections can also occur.

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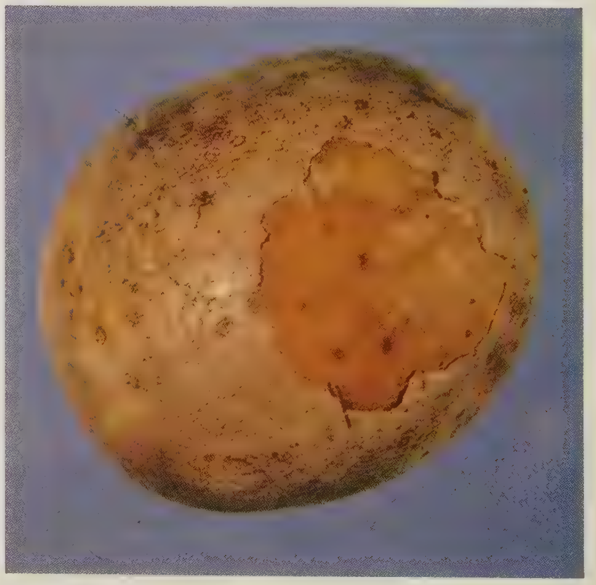

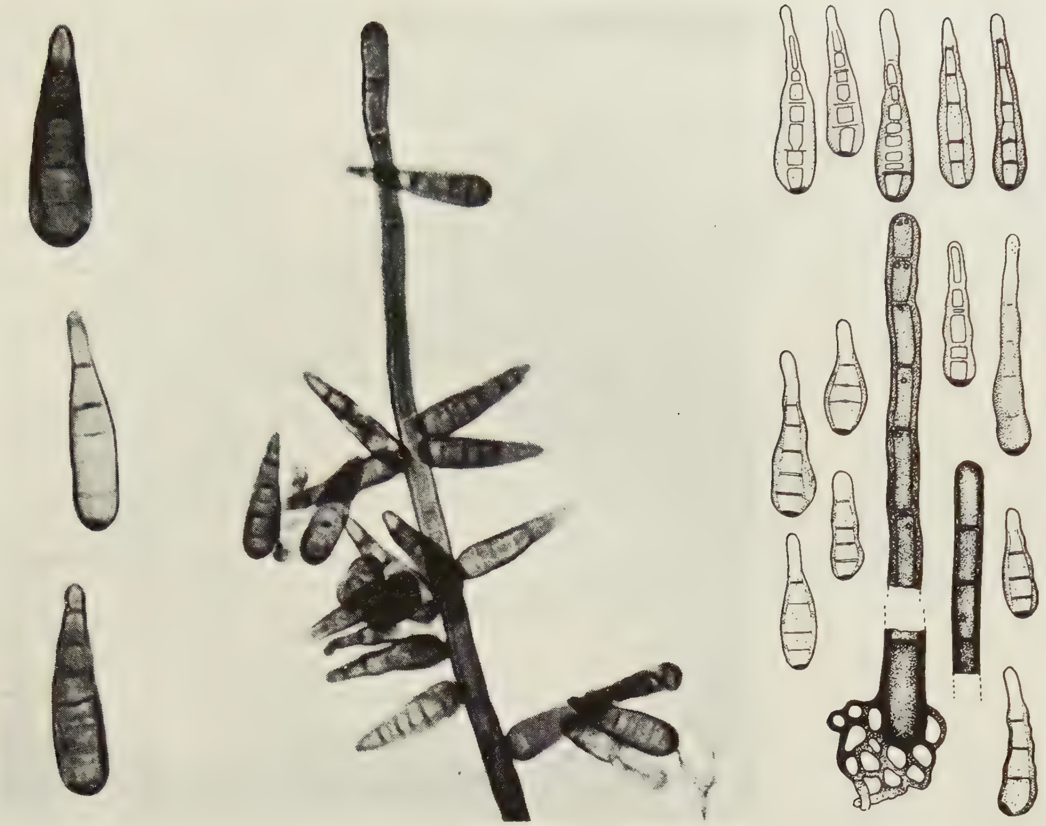

434 *Helminthosporium solani* (from CMI Descr. No. 166, Ellis 1968) Conidia and conidiophores × 500

CONTROL Crops grown from infected seed should be harvested promptly so that they may escape infection (1,4). The storage environment is very important and good air circulation is essential; after the curing period the temperature should be reduced rapidly and the relative humidity maintained below 90% (1,3,5). Infected tubers to be used as seed can be treated with a fungicide (3,4,5). Ideally, disease-free seed should be used (10), such as that obtained from stem-cuttings (3,5).

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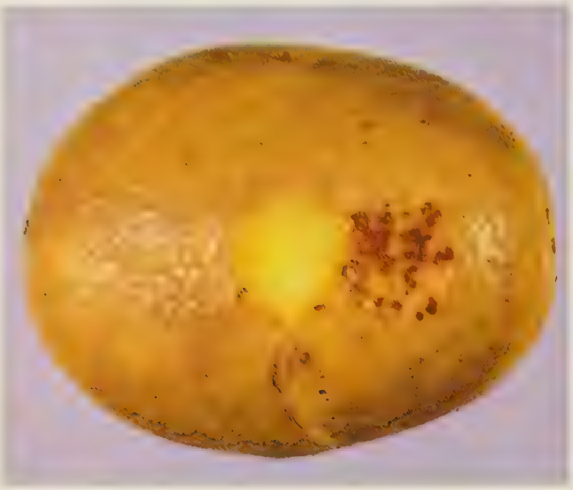
SKIN SPOT of potatoes caused by
Polyscytalum pustulans (Owen & Wakef.) M.B. Ellis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OCCURRENCE The fungus is generally restricted to cool temperate regions, and the disease appears to be of particular importance in northern European countries such as Ireland and the UK (1,2,3,4). It has also been recorded in Germany, Norway, the USSR (5), the USA, Canada, New Zealand, Australia (Tasmania) and South Africa (CMI Map 202). Skin spot constitutes a blemish which detracts from appearance in washed ware potatoes (2). The more significant aspect of the disease, however, is that it can result in death of bud tissue in the eyes, so that diseased seed tubers produce delayed and 'gappy' crops (2,4).

SYMPTOMS Small, dark, slightly raised spots develop on the skin; they may be confined to the heel end of the tuber, to damaged areas, or be distributed around and in the eyes (3). The spots are typically 1 to 2 mm in diameter and depth, though larger and deeper lesions have been reported (2). In a humid environment, spores (conidia) are produced on the surface of lesions (5).

BIOLOGY The fungus can survive in crop debris and also (as microsclerotia) in soil and on roots; in the USSR this is reported to be an important means of transmission (5). In the UK, however, it has been shown that the main source of infection is the seed (3,4). A diseased eye may fail to sprout, or may produce an infected stem (1). Spores (conidia) develop on stem-base lesions and on roots (3,4), subsequently causing infection of daughter tubers, through eyes or lenticels (1,2,5). Infection is facilitated by cold wet soil conditions (3). At lifting time abrasions may become contaminated with spores, and further infection and development occur after harvest (2). Cold (3°C) humid conditions favour spore production and spread of the fungus in store (3,4,5). The formation of cork cells (as a response to infection) restricts the expansion of skin spots. If, however, certain sprout suppressants have been applied too early, then cork formation is inhibited and lesions may be invasive (2).

CONTROL Disease-free seed, such as may be obtained from stem-cuttings, should be used if possible (3). In the USSR hygienic cultural practices are also considered important in the control of this disease, viz. crop rotation, destruction of debris, and steam disinfection of fodder potatoes before feeding to animals (since the fungus can survive passage through the digestive tract and be spread to new areas) (5). Prompt fungicide treatment of the harvested crop, by fumigation, spraying or dipping, can provide protection in store. It is possible to minimise skin spot development by manipulation of the storage environment; after the curing process the atmosphere is maintained at 10° to 15°C and 75% relative humidity (3,4,5). In practice, however, the regime chosen may have to be a compromise with requirements for control of other diseases. Potato cultivars differ substantially in their susceptibility to skin spot (1).

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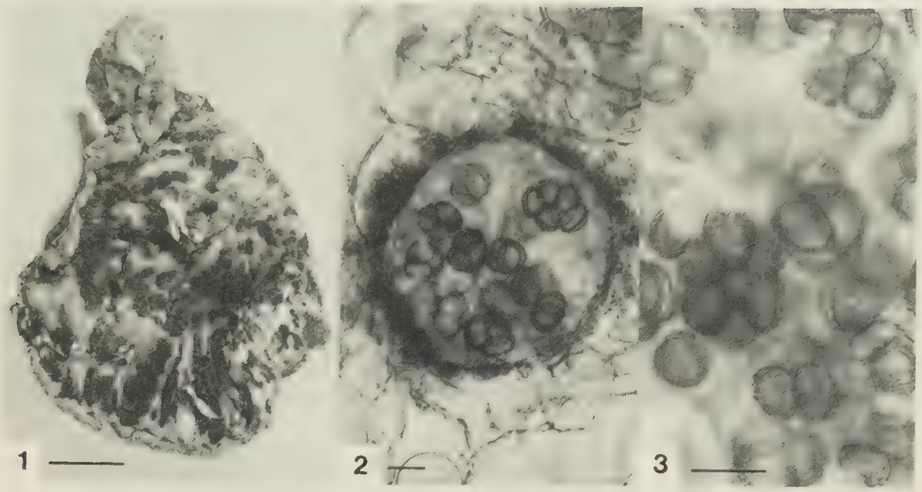
SMUT of potatoes caused by
Thecaphora solani (Thirum. & O'Brien) Mordue
(syn. *Angiosorus solani* (Barrus) Thirum. & O'Brien)



436 *Thecaphora solani* (from CMI Descr. No. 966, Mordue 1988) 1 potato tuber cut open to show internal sori 2 sorus 3 spore balls

OCCURRENCE This serious disease is presently confined to central and south American countries (CMI Map 214), including Mexico, Peru (4,5), Bolivia, Colombia, Venezuela (1,3), Ecuador and Chile (2). Heavy crop losses have been reported, both in the high Andes and in the coastal plains. Infected potatoes have been intercepted at US ports, and the fungus is a notifiable disease organism in many countries (3).

SYMPTOMS Affected tubers are malformed and, on being cut, show numerous dark brown inclusions in the flesh. These are in fact irregularly shaped cavities (sori) filled with 'spore-balls', aggregations of up to 8 individual spores (1,2,5).

BIOLOGY Spores can persist in the soil (5) and are probably distributed by rain and irrigation water. Wet soil conditions favour infection (5) and the fungus may attack stem-bases, stolons and tubers (4). Infected tissue is stimulated into cell proliferation, resulting in swollen outgrowths or tumours (1). Internal tissues are also invaded. Further deformities develop in tubers after harvest and, after several months of storage, the internal tissues may be replaced by a crumbling mass of 'spore-balls'.

CONTROL In infested areas the most effective control is to plant resistant potato cultivars, of which there are several (2,4). Chemical disinfection of the soil can be effective but is costly (4). Only clean seed tubers should be planted (5). Beneficial cultural practices include long rotation, prompt removal and destruction of infected debris, and elimination of the thorn-apple weed (*Datura stramonium*) which is also susceptible to the disease. Since the smut fungus could probably thrive in most other potato-producing countries, rigorous quarantine measures are essential (3).

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WATERY WOUND ROT or **LEAK** of potatoes caused by ***Pythium*** **spp.**

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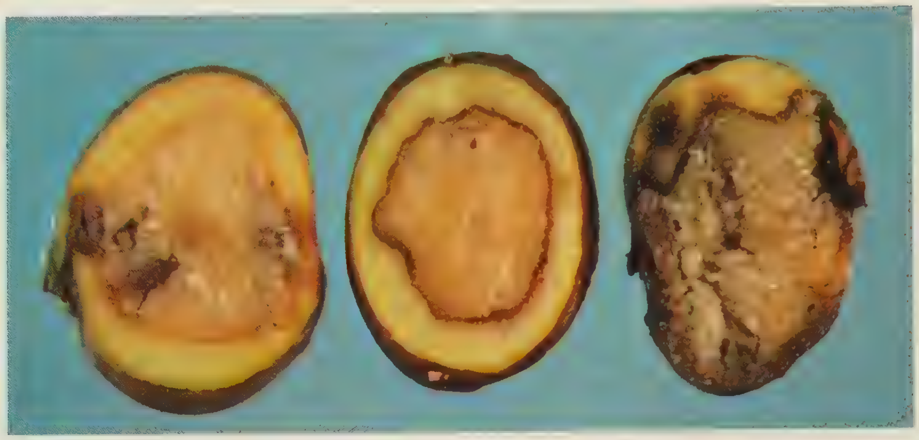

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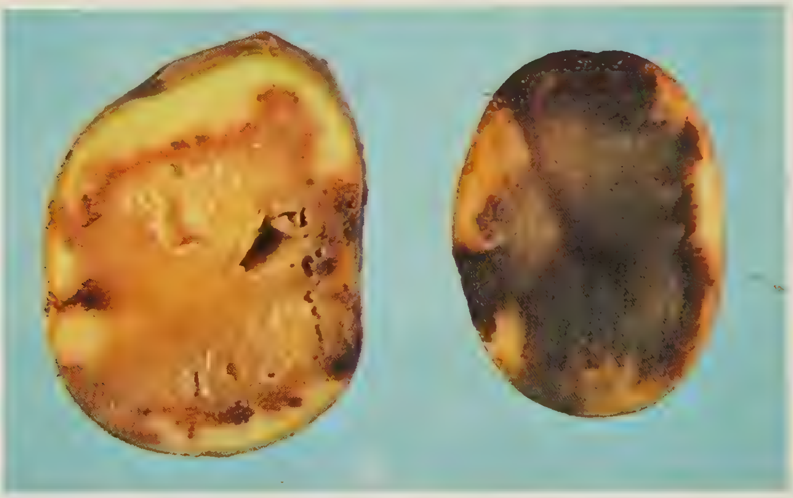
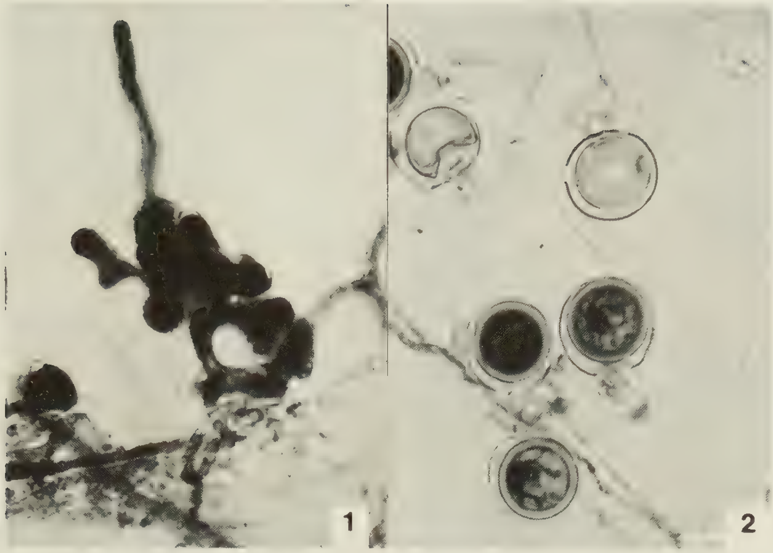


OCCURRENCE This disease is usually associated with hot weather. Records include *P. butleri* Subramaniam in India and Egypt (4), *P. debaryanum* Hesse in the USA (1,6), Australia (3) and the UK (9), and *P. ultimum* Trow in Canada (7,8), New Zealand (2), East Germany (10) and Switzerland (5).

SYMPTOMS Lesions are irregular in shape, and the affected area may be bordered by a black line, visible externally and internally (4,9). The skin is often stretched taut over the lesion owing to internal shrinkage of tissue and, if punctured, allows the release of a watery fluid (3,7). Sometimes there is little external evidence of decay yet substantial internal rotting, characteristically within the bounds of the vascular ring. The outer tissue remains firm, enclosing the fluid; if this leaks out the potato becomes hollow, hence the alternative common name **shell rot** (1).

BIOLOGY The fungi produce both a sexual stage (thick-walled oospores) and an asexual stage (sporangia); swimming spores (zoospores) may also be formed. All the species are common soil inhabitants and, particularly in wet conditions, can invade freshly cut seed pieces, causing a rapid rot (7,8). Alternatively the fungi can attack the growing plants (5), entering daughter tubers through the stem attachment (stolon) (7). More usually, however, infection takes place through injuries sustained at harvest time. Immature tubers are prone to 'scuffing' (9), while tubers of any age may sustain cuts from harvesting implements (3) or machinery, or be weakened by sun scorch. Infected tubers tend to rot early in storage (1,6) or during transport to market (2,3), and adjacent tubers become contaminated with the exuded fluid. At temperatures above 25°C infected tubers decay completely within a few days (6,7,9) and are frequently invaded by secondary bacteria (2,3). At 10°C and below, rotting is held in check (4).



439 *Pythium butleri* (from CMI Descr. No. 37, Waterhouse & Waterston 1964) 1 sporangium 2 oogonia, paragynous antheridia, and oospores × 500 (stained with cotton blue in lactophenol)

CONTROL Field hygiene is important, and discarded tubers should not be left in the field. If they are to be fed to animals, tubers should first be boiled, since otherwise the fungus may survive passage through the animal and be carried to new areas (3). Seed pieces should be treated with a fungicide (8) or they should be cured (7), so as to protect the cut surfaces from infection. At harvest time efforts should be made to minimise damage (1,2,6,9); immature tubers need especial care (3). Attention must be paid to the conditions of transport and storage. Thus, for example, stevedores should not use hooks when stowing sacks of potatoes in a ship (3), and the storage environment should be cool and dry (2,7).

---

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See also:

SOMANI A.K. (1986) *Pythium butleri* causing tuber rot of potato. *Indian Phytopathology* **39,** 529–531.

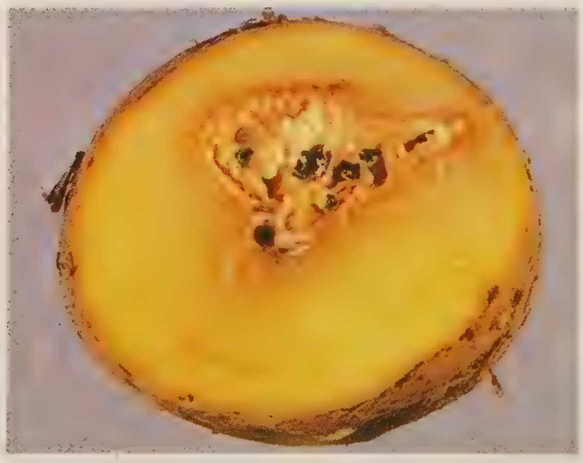
OTHER DISEASES of potatoes

BACTERIAL HARD ROT may be caused by *Erwinia carotovora* ssp. *atroseptica*. This organism can invade lenticels and wounds if conditions are sufficiently moist, and infection commonly occurs when tubers are washed (5). Subsequent storage under humid conditions results in the development of bacterial soft rot (q.v.) but, if the storage atmosphere is dry, lesions are restricted, being firm, brownish-black and sunken. Infected seed tubers give rise to plants with 'blackleg' (page 302).

CLONOSTACHYS ROT has been reported from Italy and Germany, and infection of tubers seems to be associated with wet weather around harvest time. The causal fungus is *Clonostachys araucariae* Corda var. *rosea* Preuss (4). Dark lesions develop during storage, and may support white mould bearing abundant spores (conidia).

CYLINDROCLADIUM ROT has been reported from Brazil, the causal fungus being *Cylindrocladium clavatum* Hodges & May. Infection occurs via wounds, and is favoured by warm moist conditions (1). Small brownish spots develop on the tubers, enlarging to slightly sunken circular lesions possessing a grey centre and a well-defined margin. Infected tissue is brown, dry and corky, unless subsequently colonised by bacteria which cause a moist soft rot.

GREY MOULD ROT, caused by *Botrytis cinerea* (page 281), has been recorded in the USA (7) and in Egypt. In wet field conditions the fungus can attack leaves and stems, and may invade tubers via the stolon. It also enters through wounds sustained during harvesting and handling. Immature tubers with a high moisture content are especially susceptible and undergo a brown watery decay, accompanied by wrinkling of the skin and production of grey-brown spores in the eyes. A limited dry rot has also been observed, with sunken discoloured areas. Dark resting bodies (sclerotia) may form within the tissues. Grey mould rot is favoured by cool conditions which prevent wound-healing, and may be largely controlled by ensuring that the potatoes are well cured after harvest (7).



440 Grey mould rot

NETTED SCAB, common in European countries, is characterised by superficial brown 'netted' lesions, shallower than those of common scab (q.v.). The causal organism is thought to be a species of *Streptomyces* which, in contrast to those causing common scab and russet scab (q.v.), is capable of attacking the roots of potato plants as well as the tubers (9). It is favoured by wet soils and a temperature of about 15°C.

PINK EYE, occasionally a problem in the northern USA, refers to a discoloration of the eye tissue, which turns pink and later brown. Symptoms may be apparent at harvest time in tubers lifted from wet soil, and in a humid storage atmosphere this superficial blemish can develop into a soft rot. The causal organism is a bacterium, identified as *Pseudomonas fluorescens* Migula (3). It is a weak parasite, but plants already infected with certain other organisms are predisposed to attack; thus pink eye is often associated with verticillium wilt (q.v.).

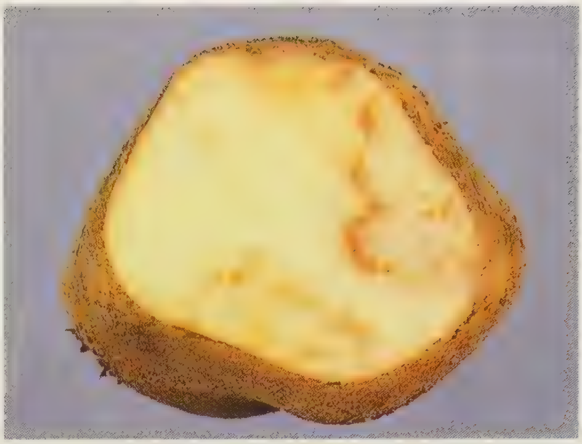
PINK MOULD ROT, caused by *Trichothecium roseum* (page 41), has been recorded in India (6). Lesions are dark and slightly sunken, and rotted tissue is typically dry, with cavities, as in fusarium rot (q.v.). Infection occurs only through wounds, and decay develops during storage. In a humid atmosphere the mould produces characteristic pale pink spores.

RHIZOPUS ROT can cause losses under warm moist storage conditions, and *Rhizopus oryzae* (page 373) has been recorded on potatoes in India. It can be controlled by dipping the tubers, as soon as possible after harvest, in a growth-regulating chemical and/or a fungicide (10). Cool storage is also effective, since the fungus makes little progress at temperatures below 15°C.

ROSELLINIA ROT has been reported from Costa Rica and other Latin American countries, where the disease is known as **torbó**. The fungus, a species of *Rosellinia*, can cause stem canker, root rot and wilt, and tubers may rot in the field or in store (8). A white fungal 'mat' envelops the tuber, darkening after a few days and giving rise to numerous aggregations of fungal strands (rhizomorphs). These penetrate the surface and grow towards, but usually do not quite reach, the centre of the tuber, which is subsequently broken down by secondary organisms. Control is achieved by improvements in drainage, destruction of crop debris and volunteer plants, together with the use of a fungicide on soil and on seed tubers. Some potato cultivars are resistant to the disease.

RUSSET SCAB, reported from the USA, is similar in appearance to netted scab (q.v.), but the causal organism appears to be incapable of attacking roots (9). Furthermore, the optimal soil temperature for disease development is about 25°C.

SCLEROTINIA ROT may be caused by *Sclerotinia minor* (7) or by *S. sclerotiorum* (2). As in grey mould rot (q.v.), decay may be soft and watery or dry and hard, and sclerotia may develop in or on the tuber. Species of *Sclerotinia* can cause a stem-rot and wilt, and infection of tubers probably occurs via the stolon. Control measures include strict field hygiene and rotation with cereals (2) (see also page 162).



441 Spraing

SPRAING is a Scottish dialect word for 'streak', and refers to brown lines in the shape of waves, arcs or complete circles, which are apparent in the flesh of affected tubers. Spraing is caused by either of two viruses, potato mop-top virus (PMTV) or tobacco rattle virus (TRV). Potato mop-top virus, named for the effect it has on the foliage, is transmitted by the powdery scab fungus (q.v.) which thrives in wet soils. By contrast, tobacco rattle virus (syn. potato corky ringspot virus) is transmitted by certain eelworms (nematodes) which tend to be prevalent in light sandy or peaty soils. Control of spraing is therefore dependent on identification and control of the vector responsible for disseminating the associated virus. Measures against the powdery scab fungus are outlined on page 320. Reduction in eelworm populations may be achieved by crop rotation and the use of chemicals, though results are not always satisfactory.

VERTICILLIUM WILT refers to a disease of the growing crop in which the fungus, *Verticillium albo-atrum* Reinke & Berthold invades the entire plant (systemic infection). Infected tubers may show vascular browning, visible as a brownish ring if the tuber is cut across at the heel end. This is not diagnostic, however, since vascular browning is also a symptom of brown rot (q.v.), ring rot (q.v.) and chemical injury caused, for example, by improper use of herbicide for 'burning off' (page 297). Verticillium wilt is controlled by rotation with cereal crops and by the use of clean or treated seed tubers. Cultivars differ greatly in susceptibility to the disease.

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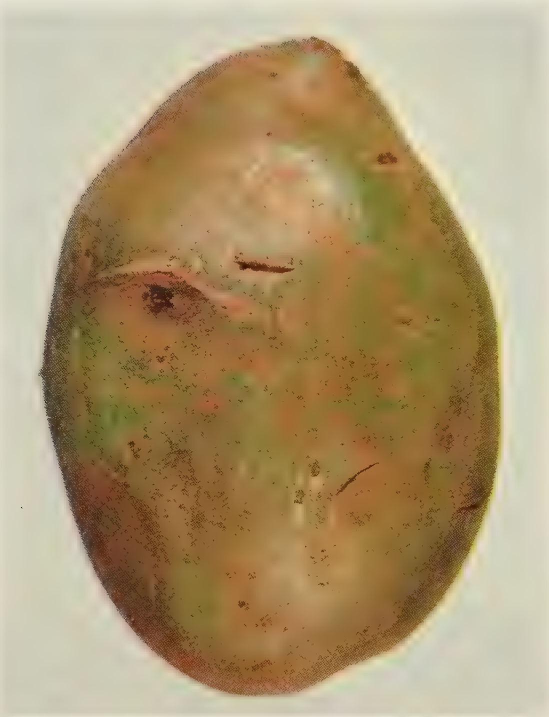
GREENING of potatoes

442



OCCURRENCE Greening, of the skin and underlying flesh, results from exposure to light. This may occur in the field if tubers project above the soil surface, and it may also occur after harvest; the risk of post-harvest greening has increased with the modern practice of displaying washed tubers in brightly lit retail shops. Investigations have been carried out in several countries, including the USA (2,3,5), the UK (1) and Australia (4).

SYMPTOMS Pre-harvest greening is characterised by an intense coloration of part of the tuber, which may have been exposed to sunlight for several weeks. In contrast, post-harvest greening (sometimes called lightburn) (3) tends to be more diffuse, often affecting the entire tuber surface. There are three components of greening, viz. external colour, internal colour, and depth of colour (5).

BIOLOGY The green pigment is chlorophyll, a normal constituent of above-ground plant parts. It is tasteless and non-toxic. There may, however, be development of other compounds (notably the glycoalkaloids such as solanine) which impart a bitter flavour and can be poisonous if consumed in large quantities (4). Susceptibility to greening varies with maturity, immature tubers being especially prone (1), and with cultivar, pale-skinned varieties generally being more susceptible than red and russet varieties (5). Temperature exerts a significant effect; one investigator found that greening developed slowly on potatoes stored at 6°C and rapidly on potatoes stored at 22°C. However, tubers stored for 2 weeks at 6°C and subsequently exposed to light at ambient temperature greened much more rapidly than those stored throughout at the higher temperature (2). Also influential is the quality (wavelength) and intensity of the light, coupled with duration of exposure. A day or two in natural light, or under fluorescent lighting, may be sufficient to cause noticeable greening. After several weeks a similar effect results even from light of very low intensity. Repeated exposure has a cumulative effect, so that potatoes which have received significant amounts of light during harvesting, handling and storage may then turn green rather rapidly in retail display.

CONTROL It is important to plant seed tubers at the correct depth, and to 'earth up' the plants when necessary. Some potato varieties tend to form their daughter tubers close to the surface, and an attack by the fungus *Rhizoctonia solani* can further aggravate this effect. After harvest, besides minimising exposure to light during harvesting and handling, care should be taken to store potatoes in complete darkness whenever feasible. Consumer preference for transparent retail packs means that exposure to light is unavoidable at this stage of marketing. Post-harvest treatment with various types of coating has proved unsatisfactory (1), and control is therefore dependent on rapid throughput of retail stock. Work is in progress to breed varieties with increased resistance to greening (5).

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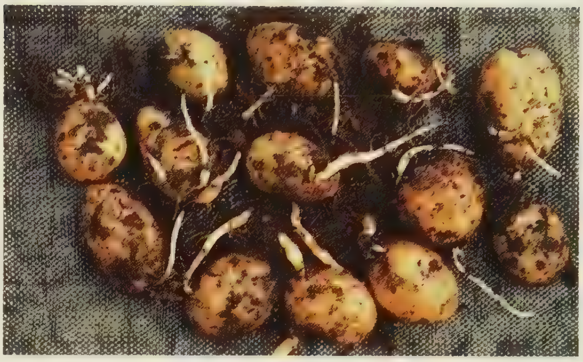
SPROUTING of potatoes

OCCURRENCE Potato tubers are storage organs with the propensity for re-growth in the form of shoots (sprouts). When tubers are used for seed, it is common practice to encourage a limited degree of sprouting ('chitting') before planting. In ware potatoes, by contrast, sprouting is undesirable and constitutes a lowering in quality; in this context it can be classified as a market disorder.

SYMPTOMS The new shoots grow out of the eyes (buds), which are located mainly at the 'rose end'. If re-growth occurs in the dark, sprouts are pale yellow, in contrast to the purple or greenish colour of sprouts formed in the light.

BIOLOGY For most potato crops there exists a period after harvest during which the tubers do not sprout even though held in an environment conducive to sprouting (6). This inherent state has been defined as 'endodormancy' to distinguish it from the dormancy which can be imposed by external storage conditions. Endodormancy commonly lasts several weeks, varying greatly with cultivar and with the conditions under which the crop was grown. In a hot dry climate, for example, some tubers may even begin to sprout in the soil *before* harvest, while others may sprout within a few days of harvest (3). Sprouting during shipment and storage is undesirable for several reasons. Moisture loss accelerates, leaving tubers soft, flabby, and with increased susceptibility to internal bruising (2). There is a marked tendency towards irreversible senescent sweetening (2). Extension of sprouts beyond about 1 cm is accompanied by a significant rise in respiratory heat production. By the time sprout growth reaches 1% by weight of a mass of tubers, respiration may have increased by 50%; if sprouting is far advanced, then the increase can be 4- or 5-fold (6). Yet another deleterious effect is the resistance to airflow as shoot growth fills up the spaces between the tubers.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CONTROL Sprouting can be held in check for as long as 8 months by storage at 4° to 5°C. The higher storage temperatures required for 'processing' potatoes (page 298) are conducive to sprouting, which must therefore be controlled by other means (1,2). Sprout suppressant chemicals are of two main types, designed either for spray application to the growing crop shortly before harvest, or for dusting or fogging of harvested tubers (1,4). Since some formulations interfere with wound-healing, it is usually necessary to defer application until completion of curing. An alternative to chemical treatment is irradiation (1), though this can result in unacceptable sweetening. A two-stage steam treatment has been recommended in India (5). The first treatment, at the onset of sprouting, kills the apical bud. A second treatment is subsequently required to kill the emerging lateral shoots, permitting storage for a further 3 to 4 months at ambient temperature. In view of the difficulties and disadvanatges of sprout suppression techniques, there is interest in breeding for long dormancy (2).

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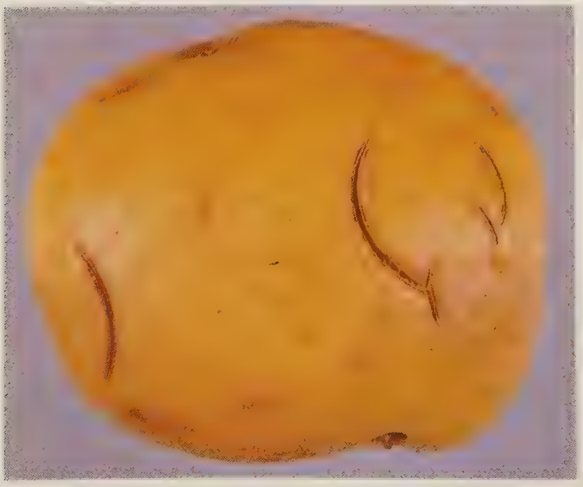
OTHER DISORDERS of potatoes

BLACK HEART is characterised by a sharply delineated, purplish-grey to black area in the centre of the tuber. Cavities may eventually develop. Only in severely affected tubers are there external symptoms in the form of moist discoloured patches. Black heart is the result of asphyxiation; if insufficient oxygen is available, normal respiration is disrupted and tissue suffers irreversible injury. This can happen in the field following flooding, or after harvest if ventilation is restricted, for example in bulk stores, ships' holds, or rail trucks (3). Oxygen starvation can also occur in conditions of good ventilation; if temperatures are excessive, the rate of respiration is so great that the demand for oxygen at the centre of the tuber cannot be met. Consequently in very hot weather potatoes should not be left in the soil after the haulm has been killed, nor should tubers be left on the soil surface at harvest. Care must be taken to provide adequate ventilation during transport and storage, so as to prevent self-heating and suffocation. Black heart may also be a symptom of chilling injury (q. v.).

CHILLING INJURY takes several forms. Discoloration may be restricted to the vascular tissue, brown flecks eventually giving way to small, dark grey blotches. Alternatively much of the flesh may show diffuse areas of grey or, in some cultivars, reddish-brown (mahogany browning). In advanced stages this may develop into black heart (4). Chilling injury is caused by temperatures below about 4°C but above the freezing point. If it is found that the store temperature has been too low, the effects may be mitigated by warming the potatoes to about 15°C for a few days and subsequently imposing a storage temperature of about 4°C (4).



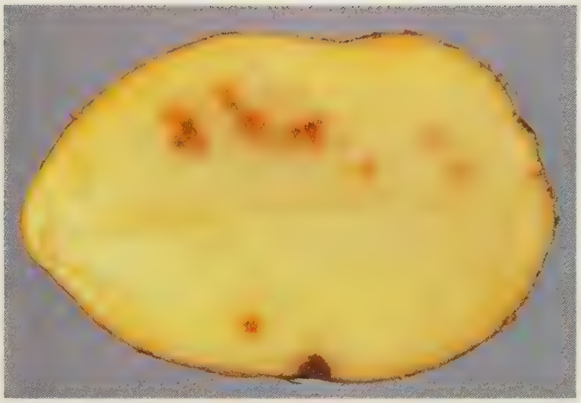
444 Thumb-nail cracking

CRACKING may take several forms, according to whether it occurs in the field or after harvest (3). Irregular water supply to the growing crop can lead to a sudden swelling of tuber tissue, followed by cracking (5). Unless invasion by micro-organisms occurs, the split tissue develops a corky layer during the normal process of wound-healing. This is unsightly in the harvested tuber, and makes peeling difficult. Cracking may also occur during harvest, if potatoes are too immature or if soil and air temperatures are too low. Tubers handled roughly after cool dry storage are especially liable to crack. Curved cracks (known as 'thumb-nail cracks' because of their shape) result from glancing impacts of tubers with other tubers or with equipment (3,7). Growth cracks can be avoided by careful attention to fertiliser and irrigation regimes. The incidence of cracks resulting from mechanical damage can be minimised by judicious choice of harvest date and time of day (avoiding very cold periods), and by warming tubers to about 15°C before grading out of store.

FREEZING INJURY can occur at temperatures below about −1.5°C, and symptoms vary with the temperature and duration of exposure (10). The vascular ring is especially liable to injury, and turns black as the tissues die (ring necrosis). The inner parts of the flesh may also be affected (net necrosis). Severe freezing results in death of the entire tuber which, on thawing, releases much liquid; such tubers are known as 'leakers'. Freezing can occur in the field or after harvest and a detailed history may be required for correct diagnosis. Observations of the pattern of damage can be invaluable. If, for example, affected potatoes appear to be dispersed at random in a load, this would be consistent with freezing (or frost damage) at an earlier stage; if, on the other hand, only the extremities of the load show symptoms, this would indicate freezing in transit (10). If undisturbed potatoes are subjected to freezing temperatures they do not necessarily freeze, but may instead undergo 'supercooling'. If supercooled tubers are allowed to warm up without being moved, they may escape injury. Alternatively, if subjected to the shock of movement while in the supercooled state, they freeze instantly.

HOLLOW HEART describes a condition in which an angular, lens-shaped or star-shaped cavity develops in the centre of the tuber (9). The cavity is cork-lined but the tissue is otherwise not discoloured. The usual cause is the sudden rapid growth which occurs if a period of drought is followed by an abundance of water. Cultivars differ in their response, but it is a general rule that large tubers are especially prone to the disorder (3). The lack of external symptoms makes it difficult to assess the incidence of this disorder, and underlines the importance of prevention by appropriate irrigation.

INTERNAL BLACK SPOT or **BLUE SPOT** is the result of bruising, either from impact during harvest and handling, or from pressure during storage (7). Within a day or two of injury, a diffuse blueish-grey spot develops in the vascular region, a few mm beneath the tuber skin, necessitating deep paring during preparation, with consequent losses. It is one of the commonest and most important defects of potatoes, and may lead to rejection of entire loads, especially in the processing industry (1). Cultivars differ in susceptibility (6), but tubers are especially vulnerable to black spot if they have previously suffered a certain amount of moisture loss (cf. cracking, q.v.). Control measures therefore include prevention of drought stress in the field, careful regulation of humidity in the store and, above all, scrupulous attention to minimising opportunities for damage (7,8). Since tubers show increased susceptibility when cold (10), it is prudent to warm them to about 15°C before grading out of store.



445 Internal rust spot

INTERNAL RUST SPOT is characterised by dry, corky, reddish-brown blotches in the flesh of the tuber, generally towards the 'rose end'. Symptoms are present at harvest and do not increase during storage; nevertheless they detract significantly from tuber quality (2). This disorder results from a localised calcium deficiency, large or long tubers being especially prone. Cultivars differ in susceptibility to internal rust spot, but a general recommendation is to ensure an adequate water supply to the growing crop, especially in hot climates where drought stress (and associated mineral deficiency) may be a problem during tuber development.

JELLY-END ROT is a physiological breakdown of the heel end of the tuber. The affected part first appears translucent or 'glassy', and later becomes soft and jelly-like. Bacteria may cause secondary breakdown or, under dry conditions, the tissue loses moisture and becomes leathery. The disorder is one of the consequences of 'second growth' of the stems and foliage, which occurs if conditions become favourable after a period of drought, excessive temperatures or other adverse factors. The sudden spurt of growth causes a withdrawal of starch from the tuber, and consequent physiological breakdown. Consignments showing a high incidence of jelly-end rot should not be stored or processed. The main preventive measure is early irrigation (3).

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Radishes

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The **radish**, *Raphanus sativus* L. (family Cruciferae), is thought to have originated in the eastern Mediterranean region, and was well-known in ancient Egypt, Assyria, Greece and Rome. Radishes of one kind or another are now grown in all temperate countries and in the highland tropics. The swollen 'root' (which includes stem tissue) is highly variable in size, shape and colour. Small spring radishes, which can be harvested within a month of sowing, are spherical or elongated, usually red, white, or red and white. There are black-skinned radishes, round or long, while others may be brown or yellowish. 'Mooli' is the name given to a kind of white radish which is allowed to grow to a length of 20 to 30 cm; some oriental radish cultivars, such as the Japanese 'daikon', can achieve an even greater size. Although the large types are generally cooked or pickled, most radishes are eaten raw, being appreciated for their crisp flesh and hot taste (which derives from mustard oil).

Harvested roots should be washed and promptly cooled. Provided that water is properly chlorinated, hydro-cooling is appropriate, and small roots can be cooled within a few minutes. Radishes may be marketed in bunches, with green tops left on; optimal holding conditions are 0°C and high humidity, so crushed ice is beneficial. If refrigeration is available, bunched radishes can be kept for 1 to 2 weeks. More usually radishes are topped and tailed; close-trimming is important in delaying the regrowth of shoots. Small salad radishes are subject to moisture loss and are therefore usually marketed in plastic film pre-packs. A slightly modified atmosphere helps to inhibit growth of both shoots and rootlets, and is preferable to the use of pre- and post-harvest chemical inhibitors. However, at moderate temperatures there is a risk of oxygen depletion, with resultant off-flavours, discoloration and soft-rot. Thus, if continuous refrigeration cannot be guaranteed, it is preferable to use either perforated or semi-permeable film.

Senescence in radishes takes the form of pithiness, in which the white flesh becomes spongy and eventually tough. Spongy roots are light in weight and may show air spaces when cut open. Post-harvest development of the disorder can be held in check by refrigeration. Care must be taken to prevent freezing (at about −1°C), since thawed roots soften as they lose water, and pigment may be lost also. Under optimal conditions, small topped and tailed radishes may be kept for up to 4 weeks, and large ones for several months.

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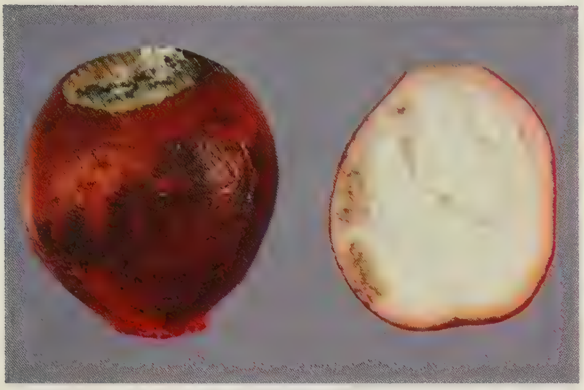
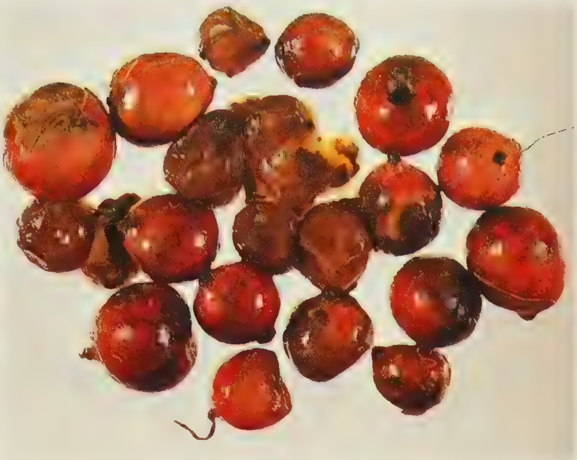


447 Downy mildew



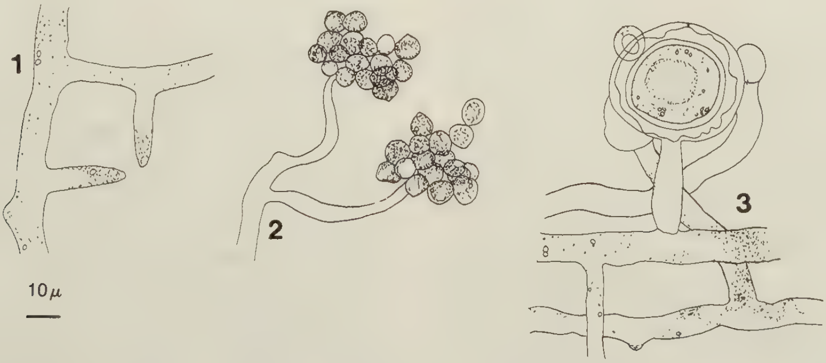
448 Bacterial soft rot

BACTERIAL BLACK SPOT is caused by *Xanthomonas campestris* pv. *vesicatoria* (page 144); serious losses of red and white radishes have occurred in the USA (9). Lesions are at first small, brown and sunken, later darkening and coalescing to cover much of the root. There is little internal discoloration. Infection occurs via injuries sustained during harvesting and handling, and symptoms can develop in packaged roots within a few days. The disease may be controlled by refrigeration but, since the 'cold chain' may not always be maintained, it is prudent first of all to chlorinate the washing water, thereby killing most of the bacteria.

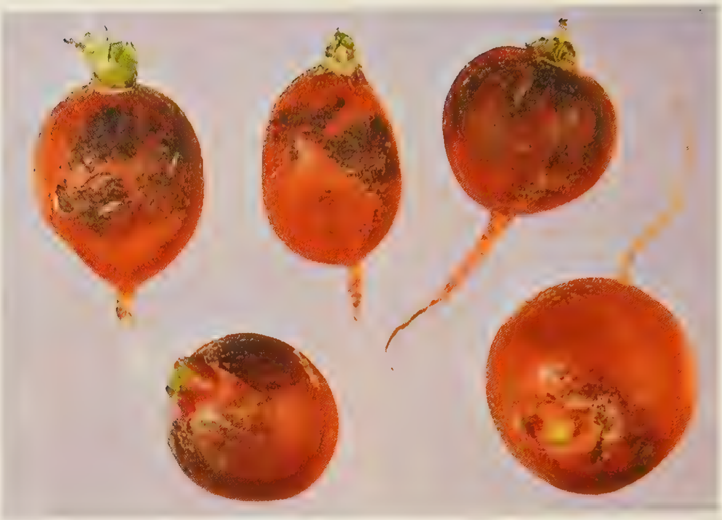
BACTERIAL SOFT ROT is caused by *Erwinia carotovora* (page 271). Infected tissue is soft, brown, slimy and malodorous, and decay extends deeply into the root. Recommendations include chlorination of the washing water, prompt cooling of the roots, and maintenance of low temperature.

BLACK ROOT ROT is especially serious in white radishes, less so in red cultivars. The causal fungus, *Aphanomyces raphani* Kendrick, survives in the soil as thick-walled oospores, and may also produce swimming spores (zoospores) (2,10). If the weather is warm and wet, radishes can be attacked at any stage of development. The result is severe stunting; the root may become constricted, and cracks sometimes appear. Infected tissue is black inside and out (4). Disease incidence can be reduced by crop rotation, avoiding other cruciferous crops such as brassicas (q.v.) which are also susceptible.

DOWNY MILDEW is one of the most important diseases of radish. Irregular black patches disfigure the shoulders and circumference of the root, and internal tissue becomes streaked with grey (8).



449 *Aphanomyces raphani* (from CMI Descr. No. 973, Hall 1989) 1 mycelium 2 sporangium with encysted primary zoospores 3 oogonium with three attached antheridia enclosing an oospore



450 Downy mildew

Sometimes the skin is russeted and cracked. The causal fungus is *Peronospora parasitica* (page 149), of which various strains exist. It survives in the soil as thick-walled oospores, which can infect seedlings via their roots. When infection progresses to above-ground parts, asexual spores (conidia) are produced in abundance on leaf and stem tissue, provided that the environment is warm and moist. These spores are dispersed in air currents, infecting the foliage of healthy radish plants or being washed down on to the developing fleshy roots. Infection of roots can occur in the field or after harvest. Direct chemical control is not possible because of the short cultivation period (1), and hence there is much interest in breeding resistant cultivars.

SCAB is caused by a strain of *Streptomyces scabies*, morphologically different from the one attacking potatoes (q.v.). Red globe radishes are more susceptible than white 'icicle' types. Symptoms are circular greyish-white lesions, with raised borders and pitted centres. Although superficial, the scabs are disfiguring and can also open the way for secondary infection. A simple method of control is to irrigate the young plants for the first 3 weeks of growth (6).

SCLEROTINIA ROT, caused by *Sclerotinia sclerotiorum* (page 47), has been recorded in India (5). Wilting occurs and the entire plant may be killed. White mould and dark resting bodies (sclerotia) develop on the fleshy roots.

SCURFY ROOT is caused by *Rhizoctonia solani* (page 306). In contrast to black root rot (q.v.), lesions are greyish brown and tend to remain superficial (4). Infected roots may become soft, wrinkled, and covered with a brownish mould (7). Control measures include soil disinfestation (7) and the breeding of resistant cultivars (4).

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Swedes (rutabagas) and turnips

The **swede**, *Brassica napus* L. var. *napobrassica* belongs in the family Cruciferae and is believed to be a hybrid between kale and turnip (q.v.). It probably originated in central Europe three or four centuries ago. In North America as well as in continental Europe swedes are known as **rutabagas**. Although considered by many to be fit only for animal fodder, swedes have a delicious taste when cooked and mashed, and are also in demand by the processing industry for pickles, soups, stews and pies. The edible part is the rounded swollen stem-base and taproot, the whole loosely called a 'root' even though much of the structure forms above the soil surface. The upper part develops a characteristic neck which eventually bears numerous leaf scars. The shoulders of the swede are purple, green or bronze in colour, while the undersides and flesh are white or, more commonly, yellowish orange.

Crop development is favoured by cool, moist weather; drought results in toughness of the root. In regions having generally mild winters, the crop can be left standing and harvested as required. Alternatively it may be covered with soil or straw *in situ*, or lifted and clamped with a straw covering. The risk of frost makes it preferable to harvest the crop before winter and, after topping and tailing, to put the roots into store. Ventilated barn storage can be satisfactory until early spring, but for long-term storage it is necessary to use refrigeration. Optimal conditions are a temperature of 0°C and a saturated atmosphere, and this is best provided by a moist-air cooling system such as the ice-bank cooler. Provided that the roots have been dipped in an appropriate fungicide, they may be cold-stored for as long as 6 to 9 months and still be acceptable to processors; surface blemishes are removed by the peeling operation. Swedes for the fresh market need to be in superior condition, and are brought out of store while still visually attractive. After the necks have been re-trimmed and any rootlets removed, the swedes are washed and surface-dried. Subsequently they may be packed in nets, or pre-packed in polyethylene bags, or else given a hot-wax treatment to minimise moisture loss and enhance appearance. Another way of marketing fresh swede is to peel it and cut it into cubes for polyethylene pre-packs. The use of perforated film results in some discoloration of the product. Sealed bags, in contrast, can prolong storage life by the creation of a modified atmosphere; this is effective only if low temperature (0° to 1°C) can be maintained, since otherwise there is a risk of off-odours. Under optimal storage conditions, hand-peeling (resulting in a smooth cut surface) confers a much longer life (36 days) than abrasion-peeling (6 days).

The **turnip**, *Brassica rapa* L., has been known in Europe since pre-historic times and is now cultivated in many countries. Young turnip tops are used as a nutritious green vegetable, but the plant is valued chiefly for its swollen 'root'. Consisting of both root and stem tissue, this storage organ differs from that of the swede in not having a neck. Turnips may be spherical, oblate or cylindrical, yellow or white, with or without a green or purple zone around the top. Some cultivars are grown for animal fodder, and others for human consumption. Principles of husbandry, harvesting, handling and storage are similar to those for swedes, except that turnips are less able to withstand frost. Traditionally turnips were bunched for market, but now it is more usual to top and tail the washed roots and pack them in nets, trays or polyethylene pre-packs. Large roots can be peeled and diced for pre-packs or for the processing industry, as described for swedes.

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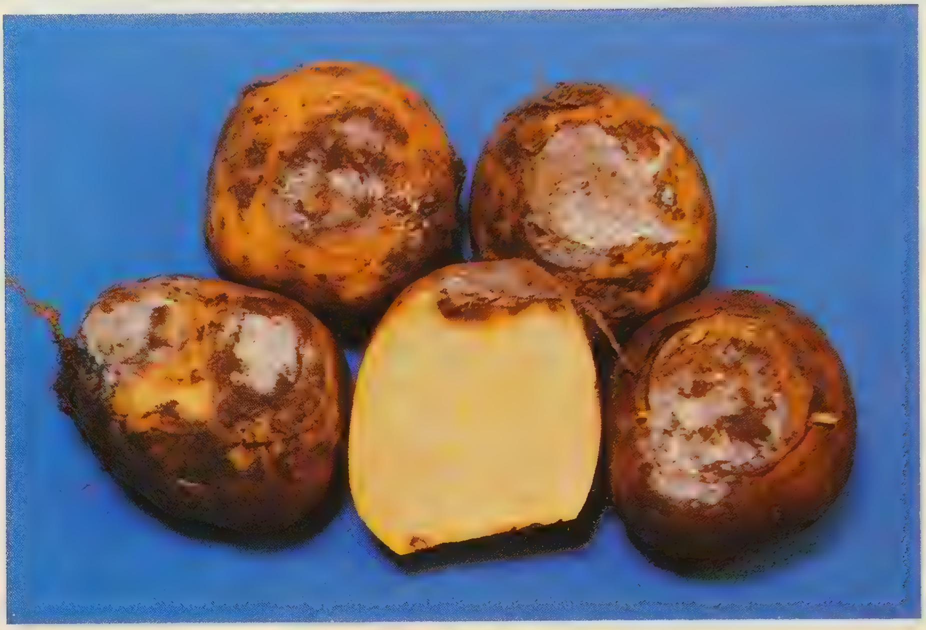
DRY ROT or **PHOMA ROT** of swedes and turnips caused by

Leptosphaeria maculans **(Desm.) Ces. & de Not.**

Conidial state: *Phoma lingam* (Tode: Fr.) Desm.

451



OCCURRENCE The disease is important in temperate and subtropical countries (CMI Map 73), including the UK (2,6), Ireland (3), Australia (8), New Zealand (1,4,5,7) and the USA (9).

SYMPTOMS Lesions tend to be of irregular shape; infected tissue becomes dry and cracked, and minute black bodies (pycnidia) are formed in abundance (2,8).

BIOLOGY Besides being harboured by cruciferous weeds (2), the fungus persists in infected brassica crop debris, on which it produces the sexual stage (pseudothecia giving rise to ascospores) (3,10). Ascospores are dispersed by wind (1) and under moist conditions can infect leaves and stems (2,8), with the formation of the asexual stage (pycnidia giving rise to conidia). These spores are waterborne and may lodge on the shoulder of the fleshy 'root'. Transmission by insects has also been demonstrated, and infection is especially likely in injured tissue (4). If the seed crop is attacked, seed may become contaminated or internally infected (8,9), leading to 'damping off' of seedlings or stem canker in the developing crop (3,6).

CONTROL Seed should be treated with a fungicide, since even a low level of seed infection can give rise to significant disease in the crop (6). It should be remembered that all types of brassicas may be susceptible, including oil-seed rape, and that spores can be blown from one field to another or carried on machinery or clothing (6,8). Separation of brassica crops in time and space is therefore advisable, and debris should be destroyed or ploughed in immediately after harvest (6,8). A post-harvest fungicide dip can be used to protect the harvested crop. There has been considerable success in the breeding of brassica cultivars resistant to this disease (6,7).

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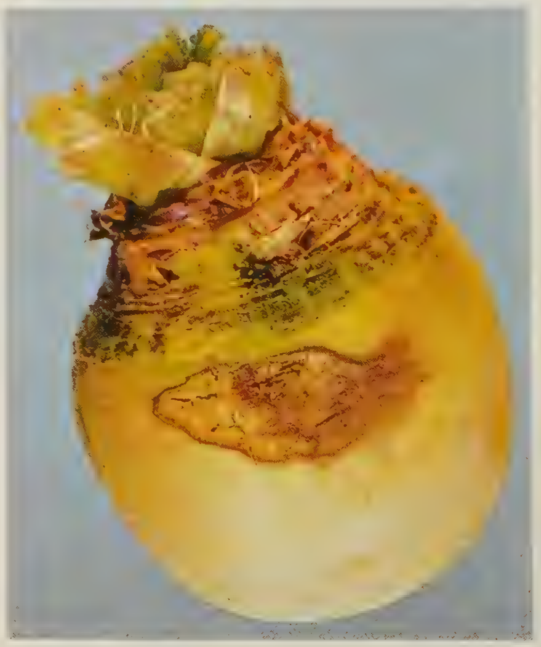

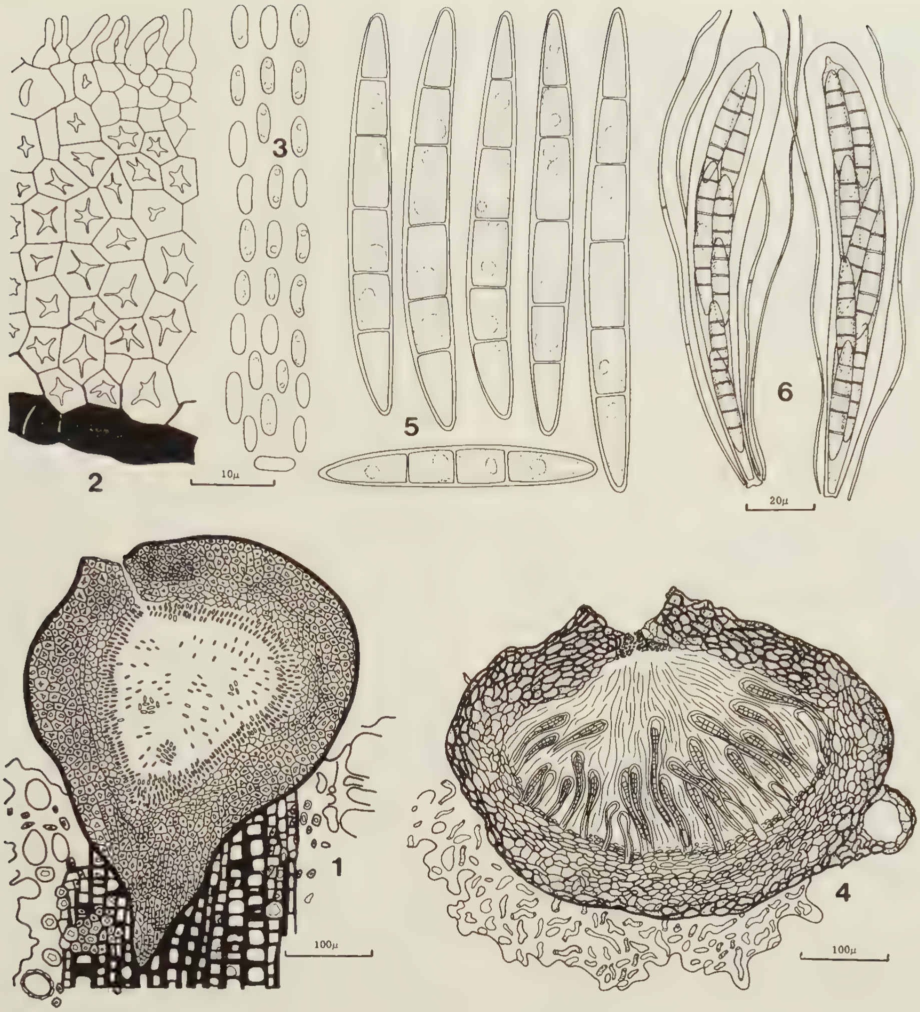

453 *Leptosphaeria maculans* (from CMI Descr. No. 331, Punithalingam & Holliday 1972)
1 pycnidium 2 part of pycnidial wall 3 conidia 4 pseudothecium 5 ascospores
6 asci and pseudoparaphyses

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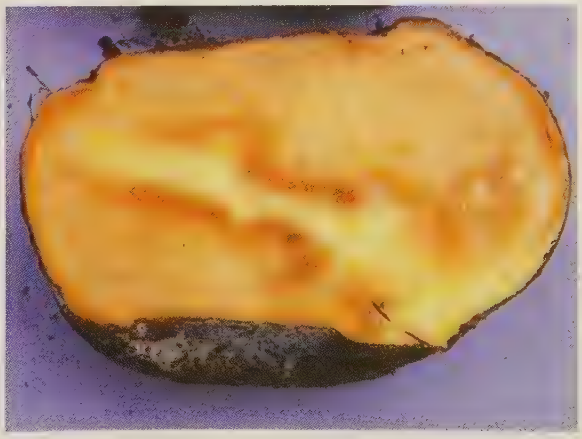
OTHER DISEASES of swedes and turnips

ALTERNARIA ROT of turnips has been recorded in the USA (2), the causal fungus being *Alternaria brassicae* (page 147). Following leaf-spotting and root-infection in the field, harvested turnips rot in store. Lesions are circular, and usually zoned in shades of brown (2). In a humid atmosphere there may be visible mould growth. The fungus is seed-borne, and it is therefore necessary to use clean or treated seed.

ANTHRACNOSE of turnips, caused by *Colletotrichum higginsianum* Sacc., has been recorded in the USA (2). Lesions are grey to light tan and markedly depressed, with slightly wrinkled borders. Moist storage conditions permit the formation of mould strands and spores (conidia); secondary bacterial soft rot often follows. Since the fungus can survive the winter in infected foliage left in the field, it is advisable to destroy all crop debris after harvest (2).

BACTERIAL SOFT ROT, caused by *Erwinia carotovora* ssp. *carotovora* (page 271), is an important field disease in many parts of the USSR (10). Bacteria in the soil invade the roots via injuries, and can cause complete rotting even before harvest. Roots with incipient infections decay in store. Chemical protection of seed is beneficial, but the chief means of control is avoidance of the disease by early harvesting, especially after a cold summer (10). Bacterial rotting of turnips may also be caused by species of *Pseudomonas; P. syringae* pv. *maculicola* has been recorded on turnips in Bulgaria (6).

BLACK ROT of swedes (8) and turnips (6) is caused by *Xanthomonas campestris* pv. *campestris* (page 144) which is seed-borne. The bacteria colonise the vascular system of growing plants (systemic infection), and harvested 'roots' may show black streaks if sectioned (8). Soft-rotting by other bacteria (e.g. *Erwinia* spp.) often ensues, especially at moderate storage temperatures. Control involves seed treatment and/or storage of 'roots' at temperatures close to 0°C (8).



454 Phytophthora rot of swede

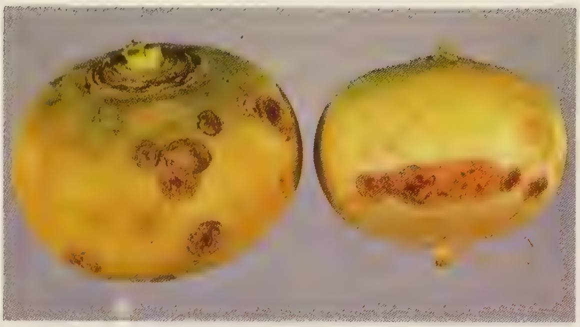
BROWN HEART of swedes and turnips is characterised by a blotchy discoloration of internal tissue, which may appear slightly watery and translucent (9). Severely affected 'roots' may have cavities in the centre, but the outermost tissues remain normal and the disorder is apparent only upon cutting. The cause is a deficiency of the trace element boron, and the problem is said to be especially prevalent after dry seasons. Incidence and severity can be reduced by the addition of appropriate quantities of borax to the soil (9), but control is not always straightforward since boron, though present, is not necessarily taken up by the plants (1). The effects of other elements, including calcium, must be taken into account.

DOWNY MILDEW of turnips has been recorded in the USA (4). Seedlings which do not succumb to 'damping off' may nevertheless harbour the fungus in the crown, from where it eventually penetrates the fleshy root. Decay is chiefly internal and may not be apparent at harvest. Affected tissue is quite firm and varies from pale brown to black, being predisposed to secondary infection by *Rhizoctonia solani* (q.v.) or bacteria. If, before this stage, the cut surface is incubated in a moist atmosphere, a fine white mould develops, bearing characteristic spores (sporangia) of *Peronospora parasitica* (page 149). The fungus probably survives the winter in turnip roots remaining in the field, and may become active the following year during mild moist weather. The disease appears to be uncommon; no control measures have been suggested, but fungicide sprays might be appropriate.

GREY MOULD ROT of turnips has been recorded in the USA and is described as a moist firm rot (7). The causal fungus is *Botrytis cinerea* (page 281) which produces characteristic grey-brown spores (conidia).

PHYTOPHTHORA ROT of swedes has been recorded in Ireland, Canada and the UK (Geeson, 1990). The causal fungus is (or is similar to) *Phytophthora megasperma* Drechsler, and sparse white mould may be present on the root surface. If an infected root is cut open, it may show extensive brown decay, spreading from the base or, less frequently, from the top of the swede and eventually affecting the entire root. Rotted tissue remains fairly firm (until invaded by secondary organisms) and typically has a slightly pungent smell. Phytophthora rot is associated with excessively wet field conditions. Control is difficult since the fungus is not affected by general-purpose fungicides, neither is it sufficiently inhibited by low temperature storage. In fields with a history of phytophthora rot, it is therefore prudent, if possible, to avoid harvesting the crop under excessively wet conditions if it is to be stored for any length of time.

RHIZOCTONIA ROT has been recorded on swedes in the UK (3) and on turnips in the USA (7). The causal fungus is *Thanatephorus cucumeris* (page 307), and the sexual state has been observed on turnip roots in the field (7). This fungus exists as several strains, and one which infects turnips does not necessarily infect potatoes, for example (7). On swedes the fungus causes a dry rot which, although fairly superficial, constitutes an important market blemish. Lesions may be sunken, shiny and brown with purplish rims. Alternatively, large irregular black craters may develop, with faint zonation and a scabby appearance (3). Swede cultivars differ in their susceptibility to this disease. On turnips the lesions are circular to elliptical, often zonate, varying from greyish brown to dark brown. Brownish mould may develop, with dark flat resting bodies (sclerotia). Internally the affected root tissue is pale brown, moist and spongy. Infection may occur in the field or during storage from soil adhering to harvested roots; mould strands invade injuries, typically at root tips, tops and broken rootlets (7). The amount of decay increases rapidly as the temperature is raised above 4°C; good control is achieved by storage at 0° to 2°C.



455 Scab of turnip

SCAB, caused by *Streptomyces scabies*, constitutes an important blemish in turnips which are washed for market. Infection, which occurs in fairly dry soil conditions, results in circular to oval outgrowths on the fleshy roots. The use of irrigation is an effective control measure (page 313).

WATERY SOFT ROT of swedes and turnips may be caused by *Sclerotinia minor* or by *S. sclerotiorum* (pages 47, 212). Records include the USA, Bermuda, the UK and Yugoslavia. Decayed tissue is wet and odourless, and in turnips a pinkish tinge may be discernible at the advancing edge of the lesion. White mould develops, giving rise to characteristic resting bodies (sclerotia). Control entails cultural methods to reduce the numbers of sclerotia in the soil. Refrigeration retards, but does not halt, the development of watery soft rot in store, so infected produce should be sorted and marketed without delay.

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See also:

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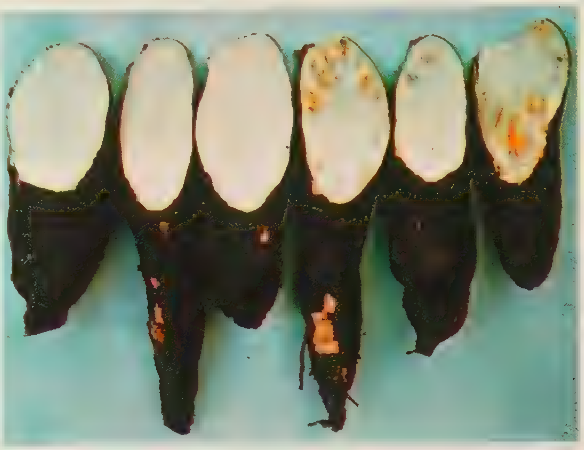
CHAPTER 9

TROPICAL ROOTS AND TUBERS



456 Vascular streaking in cassava

In much of the tropical world the root and tuber crops are important staple foods (8). **Cassava** plays a vital role in three continents (4). **Yams** are especially important in Africa (10), while **sweetpotatoes** are mostly grown in Asia (5). **Cocoyams** of various types make up a small proportion of the total but may be of great local significance, for example in the Pacific Islands (6).

They all produce starchy underground storage organs. In cassava and sweetpotatoes these are true roots, while in yams it is the stem-bases which become swollen, forming tubers. A slightly different modification of the stem occurs in cocoyams, and the resulting structures are known as corms (4,10).

It is only during the last two decades that the various 'tropical root crops' (an all-embracing term) have begun to receive the attention they deserve (4,5). Post-harvest losses are often substantial, adverse factors being the inevitability of harvest injury and the difficulties of transport and storage in a tropical environment (1,2,3,7). With the exception of cassava (which is unique in several respects), tropical roots are prone to chilling injury if held at too low a temperature (4). In practice, storage is almost exclusively at ambient temperature, and various strategies have been devised to delay the onset of deterioration (8). These techniques are described for individual commodities. Also considered in this chapter is **ginger**, of which the edible part is the swollen underground stem or rhizome (9). In complete contrast to the staples, ginger is consumed in very small quantities as a flavouring or as a spice. In its post-harvest properties, however, it has much in common with the 'tropical roots'.

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See also:

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Cassava

Cassava, *Manihot esculenta* Crantz, has a special significance in world food supplies. Although the roots are low in protein (10) and even potentially toxic (2), carbohydrate yields are high on marginal land which may produce little else (3). Furthermore, the mature roots can be left unharvested for a considerable period of time. This makes cassava a valuable standby in countries where drought sometimes precludes the production of other crops (10). More than that, in normal circumstances it provides food for many millions of people in Africa, Asia and South America (3,4,7). World production is approximately 140 million tonnes, the top ten producers being Brazil, Thailand, Zaire, Nigeria, Indonesia, Tanzania, Uganda, India, Paraguay and Mozambique. A proportion of the total is processed for animal feed, while some goes to manufacture starch, of which tapioca is one of the edible forms. In some countries, for example Thailand, the crop is grown chiefly for export in processed form.

Cassava was cultivated in antiquity in tropical America, and subsequently spread from the mainland to the Caribbean islands (3). In the 16th century the Portuguese introduced it into West Africa. Some two centuries later it entered East Africa (via Madagascar), and at about the same time also reached Asia. It is now grown throughout the tropics.

The cassava plant is a perennial dicotyledonous shrub belonging to the family Euphorbiaceae. It is propagated by means of stem-cuttings, an advantage in that the entire yield of roots can be utilised productively. Cassava is consumed in many forms, boiled as a vegetable, or pounded and fermented, or sliced, dried and milled into flour (3). The cooking and processing are necessary to reduce the naturally occurring cyanide (prussic acid) which is produced when cells of the root tissue are ruptured (2). Chronic cyanide poisoning may occur in circumstances where cassava forms a major part of the diet, but appears to be entirely prevented by adequate intake of protein from other sources (3).

Fresh cassava is generally consumed close to the place of production (4), of necessity since it is uniquely perishable, with the potential for serious physiological deterioration within 24 hours of harvest (8). This primary deterioration takes the form of vascular streaking (q.v.), later followed by secondary microbial deterioration caused by various fungi and bacteria (6,8). Post-harvest characteristics can be modified by pre-harvest practices; in particular, if the aerial part of the plant is drastically pruned 2 or 3 weeks before harvest, then the roots show a reduced tendency to deteriorate (10). It is also advantageous to leave a short 'stub' of stem attached to the harvested roots, thereby minimising injury (10).

Curing at high humidity improves potential storage life, and a moist storage environment is essential to control primary deterioration (3,5). Packing the roots in damp sand or sawdust can be effective for short periods (5). More practically, deterioration can be delayed by the simple expedient of putting the harvested roots promptly into polyethylene bags (9). If the roots are first dipped in a fungicide to control microbial spoilage, then they can be kept for 2 weeks or more at ambient temperature (1,4,5). This extension of storage life is sufficient to permit not only orderly marketing locally but also exportation, for example from South America to the USA (1). Alternative techniques include waxing to minimise moisture loss (5), and refrigeration to slow down both physiological and pathological processes. Alone amongst the tropical root crops cassava tolerates low temperatures, and can be stored at 0° to 2°C for as long as 6 months (10).

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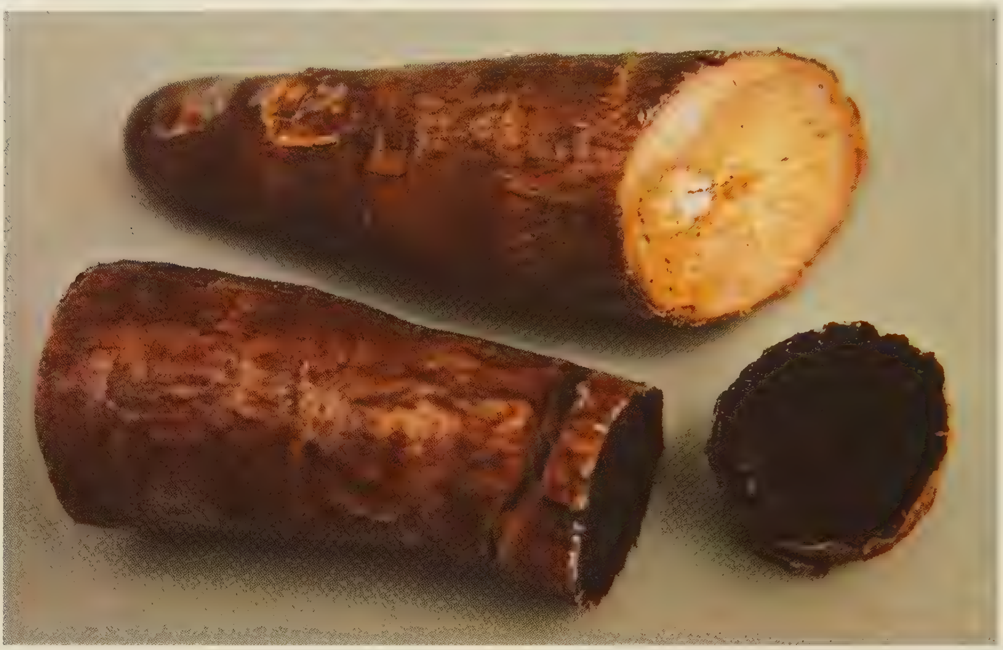
BOTRYODIPLODIA ROT of cassava caused by
Botryodiplodia theobromae **Pat.**

457



OCCURRENCE This disease has been recorded in Colombia, Costa Rica, Brazil (2,3), Bermuda (4), Nigeria (1) and India (5), and probably occurs wherever cassava is grown.

SYMPTOMS Infected roots may appear healthy externally (1), although the skin may be somewhat wrinkled (2). The internal tissue is discoloured and, under humid conditions, a fine white (later dark grey) mould develops on the cut surface (1). If infected roots are stored for some weeks they may become mummified (3); in and beneath the skin are numerous minute bodies (pycnidia) which discharge their spores (conidia) in sticky 'tendrils'.

BIOLOGY The fungus survives in crop debris, and pycnidia have been found on moribund aerial parts of stems (4). The organism may be transmitted to new areas in infected planting material. The spores are dispersed by rain or irrigation water, and can cause infection of injured tissue. Stormy weather can predispose the plants to infection (4). Stems may be infected via cracks caused by wind damage, while roots may be invaded before harvest via growth cracks or at harvest through the inevitable cuts sustained when the roots are severed (1,2,4). Rotting is rapid at tropical temperatures (1).

CONTROL Of ten cassava cultivars assessed in Brazil, all were susceptible to the disease (3). Decay can be controlled by low temperature storage or by the use of a fungicide treatment applied immediately after harvest (1,5).

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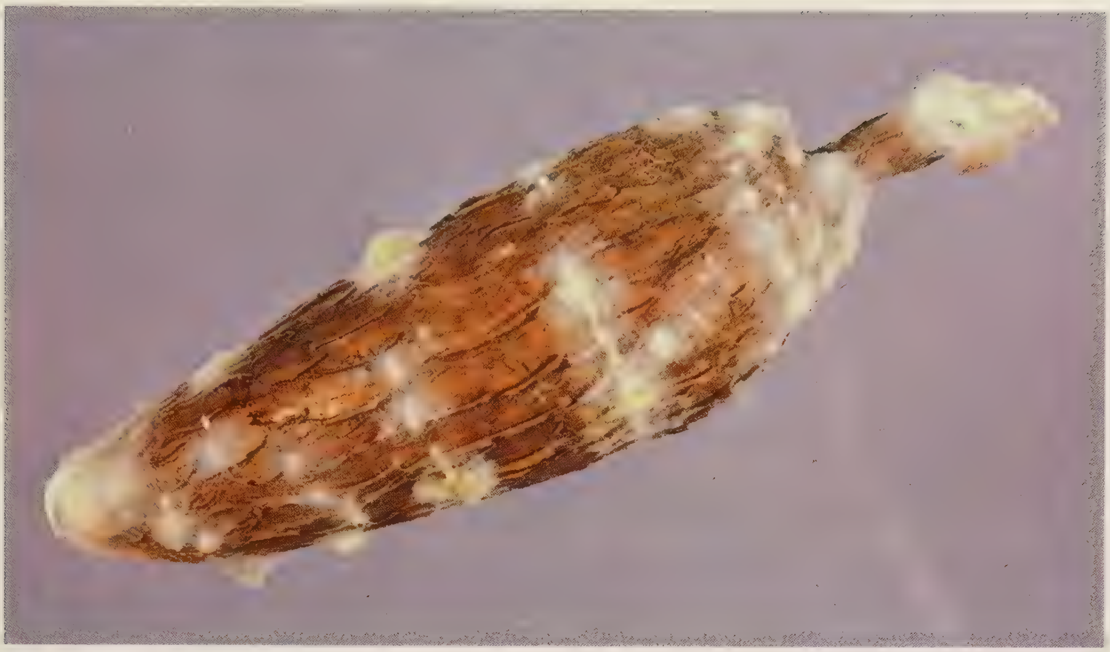
4 SEAVER F.J. & WATERSTON J.M. (1942) Contributions to the flora of Bermuda. 3. *Mycologia* **34,** 515–524.

5 SIVAPRAKASAM K., KRISHNAMOHAN G. & KANDASWAMY T.K. (1976) Evaluation of fungicides for the control of sett rot of tapioca caused by *Diplodia natalensis. Madras Agricultural Journal* **63,** 424–425.

FUSARIUM ROT of cassava caused by *Fusarium solani* (Mart.) Sacc.



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OCCURRENCE Fusarium rot has been recorded in Nigeria (3), India (4,5), Costa Rica (2), Colombia (1) and in cassava imported into the USA from Puerto Rico.

SYMPTOMS A dry rot is characteristic, with little external evidence of disease, although in a humid atmosphere there may be white mould growth on the surface. Infected flesh is discoloured brown.

BIOLOGY The fungus can survive in the soil as thick-walled resting spores (chlamydospores). Under favourable conditions it produces abundant asexual spores (conidia) (page 315) which are dispersed by wind and water (4). Infection of roots may take place before or during harvest (5), and is typically via injuries. Rotting is moderately rapid in comparison with that caused by other fungi (3).

CONTROL Since this fungus can make substantial progress even at low temperatures, the chief means of control is the use of a post-harvest fungicide dip (3,4).

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1 BOOTH R.H. (1978) A review of root rot diseases in cassava. In *Proceedings of the 1977 Cassava Protection Workshop*, pp. 121–133. Cali, Colombia: Centro Internacional de Agricultura Tropical.

2 CABALLERO Ll., C. & VALVERDE G., E. (1983) Evaluación de métodos para el almacenamiento y conservación posterior a la cosecha de raices de yuca, cv. Valencia. *Agronomia Costarricense* **7**(1/2), 55–61.

3 IKEDIUGWU F.E.O. & EJALE A.U. (1980) Root-surface mycoflora of cassava (*Manihot esculenta*) and post harvest rot of the tubers. *Mycopathologia* **71**, 67–71.

4 MISHRA D. & RATH G.C. (1986) Some pathological aspects of *Fusarium solani* (Mart.) Sacc. causing postharvest decay of *Colocasia* and yam. *Indian Journal of Mycology and Plant Pathology* **16**, 323–325.

5 SIVAPRAKASAM K., KRISHNAMOHAN G., RAMAKRISHNAN G. & SUBRAMANIAN C.L. (1977) A new dry rot of tapioca tuber incited by *Fusarium solani*. *Madras Agricultural Journal* **64**, 208–209.

OTHER DISEASES of cassava

ASPERGILLUS ROT may be caused by various species, for example *Aspergillus flavus* has been recorded in Colombia, and both *A. flavus* and *A. niger* in Nigeria and India. Roots are contaminated at harvest, and infection takes place via wounds. Post-harvest decay is favoured by warm humid conditions, which encourage the development of abundant spores (conidia) on the cut surface; those of *A. flavus* are yellowish or greenish, while those of *A. niger* are dark brown or black (page 243).

BACTERIAL SOFT ROT is probably quite common. Primary rotting may be caused by *Erwinia carotovora* ssp. *carotovora* (page 271) (2). Other bacteria, for example *Bacillus subtilis*, may follow fungal infections, resulting in a soft wet evil-smelling decay (5). Bacterial soft rot is encouraged by warm moist conditions and poor ventilation.

MUCOR ROT, caused by *Mucŏr hiemalis*, has been recorded in India (5), and a similar disease occurs in Colombia. The fungus induces a soft rot, and there may be luxuriant mould growth with globular spore heads which darken as they mature. Control measures include careful handling of the roots in order to minimise injury (1).

PHYTOPHTHORA ROT may affect cassava roots whilst they are still in the ground (4), and decay is likely to continue after harvest. *Phytophthora cryptogea* and *P. erythroseptica* (page 319) have been recorded on cassava in Zaire, and *P. drechsleri* (page 39) has been reported from Brazil and Colombia (4). Waterlogged soils predispose roots to infection, and even uninjured roots are susceptible. Rotting is rapid at temperatures between 20° and 30°C.

RHIZOPUS ROT is frequently reported as the main type of cassava decay in India (5). Rotting is rapid, and there may be copious production of spore-heads (sporangia) which are white at first, later black. The causal organism is *Rhizopus oryzae* (page 373), and it gains entry through wounds sustained at harvest, or through cracks which develop as the skin dries during storage. Control therefore entails minimising damage to the roots (1), and preventing dehydration by storing them in pits in alternate layers of moist sand, sawdust and soil.

SCLEROTIUM ROT, caused by *Corticium rolfsii* (page 255), may affect roots before and after harvest, predominantly during the dry season. Abundant spherical resting bodies (sclerotia) are produced, 1 to 2 mm in diameter, turning from white to dark brown as they mature. The fungus is soil-borne and difficult to eradicate. Infected plants should be removed and destroyed. Post-harvest rotting can be completely controlled by low temperature storage.

SPHAEROSTILBE ROT has been recorded in the Congo; it is described as a dry rot and may already be apparent at harvest. Internal tissue appears 'marbled', and after some time in store the entire root becomes mummified and covered with spore-bearing bodies (coremia) of the fungus *Sphaerostilbe repens* Berk. & Br. Its spores (conidia) are dispersed by water, and the disease is associated with badly drained soils (3). Affected plants, which may show abnormal drying of the leaves, should be removed and destroyed.

TRICHODERMA ROT can cause substantial losses; the disease has been recorded in Colombia, Costa Rica and Nigeria, and is caused by *Trichoderma harzianum* Rifai. Characteristic dark green spores (conidia) are produced on the cut surface of infected tissue (1).

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1 BURTON C.L. (1970) Diseases of tropical vegetables on the Chicago market. *Tropical Agriculture* **47**, 303–313.

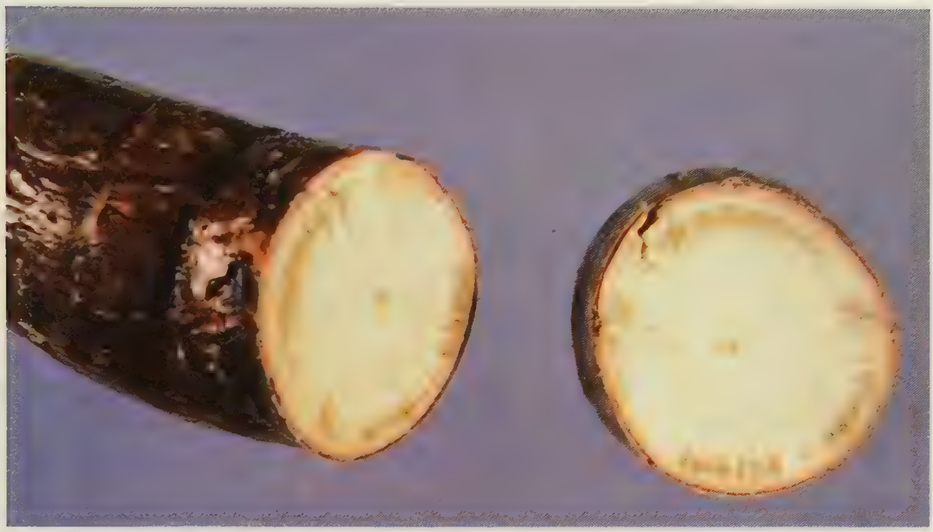
2 DANIEL J.-F., BOHER B. & KOHLER F. (1981) Les maladies bactériennes du manioc (*Manihot esculenta* Crantz) en République Populaire du Congo et en République Centrafricaine. *Agronomie* **1**, 751–757.

3 MAKAMBILA C. & BAKALA L. (1986) Les pourridiés à *Armillaria* sp., *Sphaerostilbe repens* B. et Br. et *Phaeolus manihotis* Heim sur le manioc (*Manihot esculenta* Crantz). *Agronomie Tropicale* **41**, 258–264 illus.

4 OLIVEROS B., LOZANO J.C. & BOOTH R.H. (1974) A *Phytophthora* root rot of cassava in Colombia. *Plant Disease Reporter* **58**, 703–705 illus.

5 PHILIP S. (1981) Sequence of occurrence of microflora in stored cassava tubers. *Agricultural Research Journal of Kerala* **19**(1), 126–127.

VASCULAR STREAKING of cassava



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OCCURRENCE Vascular discoloration is the typical primary deterioration (physiological in nature) shown by most cassava cultivars wherever they are grown (1,3).

SYMPTOMS If the root is cut transversely, a blue-black discoloration is apparent in the ring of vascular tissue (3). In longitudinal sections of roots the disorder takes the form of streaks, and has been described as 'blue vein' (4). Cooking quality and taste are impaired.

BIOLOGY Cassava roots characteristically deteriorate with great rapidity after harvest, as a result of complex biochemical changes following wounding (4,5). Discoloration can occur within 24 hours at 35°C and within 48 hours at 25°C. It is supposed that the disorder occurs as a result of stress produced by high rates of water loss from cuts and abrasions (2,5). Physiological deterioration is accompanied by increases in respiration rate and ethylene production (2).

CONTROL To prolong root storage life of susceptible cultivars, a simple and effective strategy consists of pruning the aerial parts of the plant 2 or 3 weeks before harvest (2). At harvest it is advantageous to leave a short 'stub' of stem attached, so that the root itself does not need to be cut. Discoloration can be wholly or partly inhibited by heating the roots for a short period before storage, or alternatively by keeping them immersed in water (2). More practical methods include thorough curing (page 345), cold storage, and dipping roots in wax as a means of minimising moisture loss. High humidity storage in polyethylene bags is especially beneficial, and permits successful storage for several weeks; a fungicide dip serves to prevent decay. Cassava cultivars differ greatly in susceptibility to vascular streaking, and some are highly resistant (3).

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1 BOOTH R.H. (1976) Storage of fresh cassava (*Manihot esculenta*). 1. Post-harvest deterioration and its control. *Experimental Agriculture* **12,** 103–111.

2 MARRIOTT J., BEEN B.O. & PERKINS C. (1978) The aetiology of vascular discoloration in cassava roots after harvesting: association with water loss from wounds. *Physiologia Plantarum* **44,** 38–42.

3 MONTALDO A. (1978) Vascular streaking of cassava root tubers. *Tropical Science* **15,** 39–46 illus.

4 PACHECO J.A. de C. (1952) Alterações de qualidade da fécula durante o almacenamento das raízes de mandioca. *Bragantia* **12,** 297–298.

5 RICKARD J.E. (1985) Physiological deterioration of cassava roots. *Journal of the Science of Food and Agriculture* **36,** 167–176 illus.

See also:

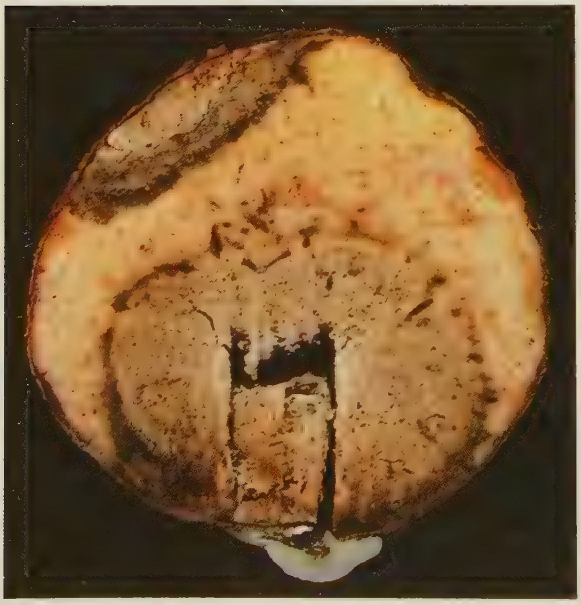
HIROSE S. (1986) Physiological studies on postharvest deterioration of cassava roots. *Japan Agricultural Research Quarterly* **19,** 241–252.

Cocoyams

Cocoyams are also known as edible aroids, being members of the monocotyledonous family Araceae. They possess very large heart-shaped leaves, and produce corms as a result of secondary thickening of the stem-base beneath the ground. Cocoyams can give high yields in parts of the humid tropics, and in several countries they constitute an important item in the diet (1,2,6,8).

Taro, *Colocasia esculenta* (L.) Schott, is thought to have originated in India. In very early times it spread eastwards into Malaya, China, Japan and the Pacific Islands, and westwards to Egypt and other parts of Africa (10). Taro has been grown in West Africa for perhaps two thousand years, and this long-established crop is known there as 'old cocoyam' to distinguish it from more recent arrivals (2). Not until the 18th century did taro reach the New World, but it is now a popular crop in the West Indies (9). World production is of the order of 6 million tonnes, the top ten producers being Nigeria, China, Ghana, Japan, the Ivory Coast, Papua New Guinea, Burundi, the Philippines, Egypt and Madagascar. It is of considerable importance in the South Pacific Islands (5) and in Hawaii (10).

Taro is tolerant of flooding, and indeed gives higher yields when cultivated in standing water (10). Two main types are usually recognised, distinguished by the ways in which they produce their corms. In *Colocasia esculenta* var. *esculenta*, known as dasheen, there is a large edible main corm (cylindrical, about 30 x 15 cm) to which are attached a few lateral 'cormels'. In *C. esculenta* var. *antiquorum*, known as eddoe, it is the numerous cormels which are edible, the main corm being small and bitter. Confusingly, eddoe has sometimes been called dasheen (3). Recently a third type (a natural mutant) has been described, in which the corms and cormels are not distinct but form a conglomerate (7).



460 Aspergillus rot of tannia (by inoculation)

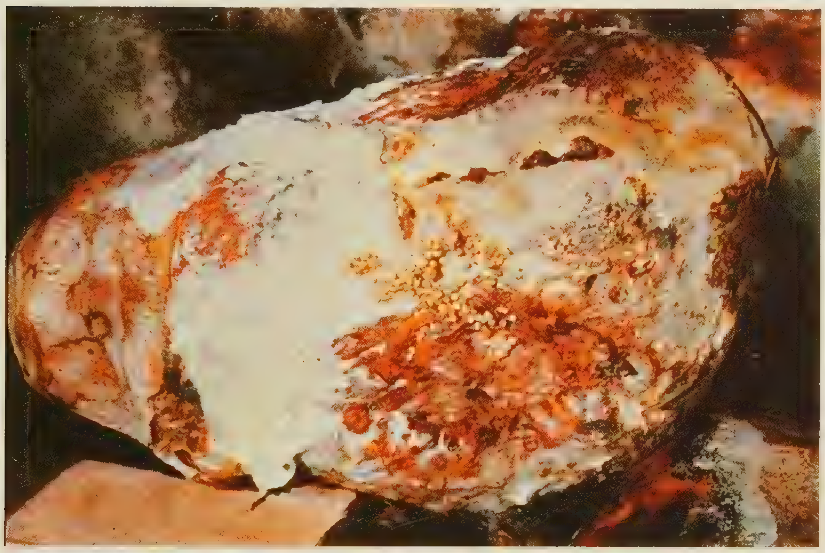
Dasheen and eddoe differ in their post-harvest characteristics, a reflection of their differing structures. The harvested corm of dasheen is left with several wounds after removal of the cormels, and suffers further injury if the basal portion is cut off to provide planting material. The remaining corm is thus predisposed to fungal invasion on several fronts. In contrast, the cormels of eddoe each bear only one wound, following separation from the mother corm. Furthermore, the cormels of certain cultivars of eddoe possess some degree of dormancy, which confers additional storage life. Thus, while eddoe may be stored for up to 3 months at ambient temperature, dasheen is more perishable; even in a warm moist storage environment (which hastens curing of wounds), the corms cannot usually be held for longer than about a month without undergoing sprouting and decay. Some reports indicate a very much shorter post-harvest life for both dasheen and eddoe (4,9). Post-harvest treatments can be beneficial, choice of fungicide being dictated by the predominant decay organisms (4). A temperature between 11° and 13°C can extend storage life significantly. Taro is sometimes shipped at temperatures substantially lower than this, and must be consumed within a day or two of release from refrigeration. Taro is nutritionally superior to other tropical roots and tubers, and also its starch is very fine-grained, making it easily digestible (2).

Tannia, *Xanthosoma sagittifolium* (L.) Schott, was first cultivated by the Amerindians, and has been grown for thousands of years in tropical America and the Caribbean islands (1,2,6). Only in the 19th century was it introduced into Africa, where it became known as 'new cocoyam' to distinguish it from the indigenous taro (q.v.). Europeans took tannia to the Pacific Islands, where it is sometimes called European taro.

Xanthosoma is a more robust plant than *Colocasia*, and is distinguished by the shape of its leaves (6). The crop can be grown without irrigation, and is comparatively resistant to pests and diseases. The corm of tannia is roughly spherical, and its cormels are flask-shaped and quite large (15 to 25 cm in length). They have a natural dormancy and may be kept for up to 2 or 3 months in well-ventilated surroundings. Sometimes ashes are used to cover the cormels, and this may have a fungicidal effect. As with taro, methods of cooking include boiling, baking, roasting and frying, and there are several methods of processing (1,2).

SAMPLE
14 LB

461 Part of trial shipment of cocoyams (see below)



462 Sclerotium rot of sample cocoyams

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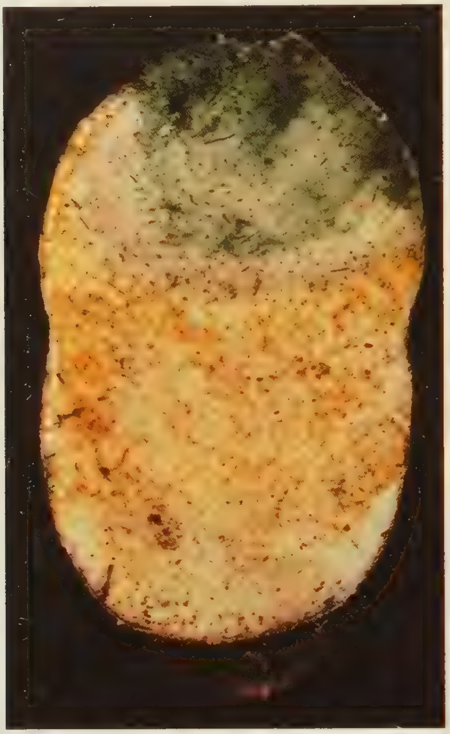
1 CHANDRA S. (1984) *Edible aroids.* Oxford: Clarendon Press, 252 pp.
2 COURSEY D.G. (1968) The edible aroids. *World Crops* **20**(4), 25–30 illus.
3 HODGE W.H. (1954) The dasheen, a tropical root crop for the south. *Circular of the United States Department of Agriculture* No. 950, 28 pp.
4 JACKSON G.V.H. (1980) *Diseases and pests of taro.* Noumea, New Caledonia: South Pacific Commission, 51 pp illus.
5 LAMBERT M. (1982) *Taro cultivation in the South Pacific.* Noumea, New Caledonia: South Pacific Commission, 144 pp illus.
6 O'HAIR S.K. & ASOKAN M.P. (1986) Edible aroids: botany and horticulture. *Horticultural Reviews* **8,** 43–99.
7 OKONKWO C.A.C. (1987) Polymorphism in taro (*Colocasia esculenta* (L.) Schott) and its implication in taro breeding and production in Nigeria. *Journal of Horticultural Science* **62,** 395–400 illus.
8 PANCHO J.V. (1959) Notes on cultivated aroids in the Philippines: the edible species. *Baileya* **7,** 63–70.
9 PASSAM H.C. (1982) Experiments on the storage of eddoes and tannias (*Colocasia* and *Xanthosoma* spp.) under tropical ambient conditions. *Tropical Science* **24,** 39–46.
10 WANG J.-K. & HIGA S. (Eds.) (1985) *Taro: a review of* Colocasia esculenta *and its potentials.* Honolulu: University of Hawaii Press, 400 pp illus.

BOTRYODIPLODIA ROT of cocoyams (taros and tannias) caused by ***Botryodiplodia theobromae*** Pat.

463



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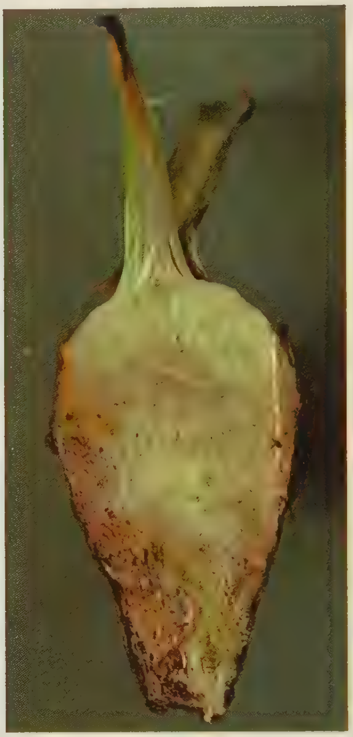

OCCURRENCE This disease appears to be quite common, and has been reported from the southern USA (3), Nigeria (1), India (4,5) and islands of the South Pacific (2).

SYMPTOMS There may be no external symptoms even when decay is advanced. Internal tissue is at first greyish or pinkish, later darkening to blue-black with an indistinct margin (2,3). Its texture may be spongy (2) and the disease has been described as a dry rot (5), but in the presence of secondary organisms a wet rot may develop. Minute bodies (pycnidia) develop beneath the skin (3) and eventually spores (conidia) ooze out on to the surface (page 371).

BIOLOGY The fungus is common in tropical soils, and may attack developing corms (4). More usually, however, corms are simply contaminated at harvest (with soil-borne or air-borne spores) and infection occurs via wounds sustained during handling (2,3). Serious decay can occur within a few days at tropical temperatures (2).

CONTROL Curing the corms (page 350) is beneficial (2), since this permits healing of wounds which might otherwise provide a means of infection (3). Fungicide treatment can give satisfactory control if cool storage is not available (1).

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1 FAJOLA A.O. & NWUFO M.I. (1985) Control of corm rots of cocoyam (*Colocasia esculenta*) caused by *Botryodiplodia theobromae* and *Sclerotium rolfsii*. *Fitopatologia Brasileira* **10**, 49–56.

2 GOLLIFER D.E. & BOOTH R.H. (1973) Storage losses of taro corms in the British Solomon Islands Protectorate. *Annals of Applied Biology* **73**, 349–356 illus.

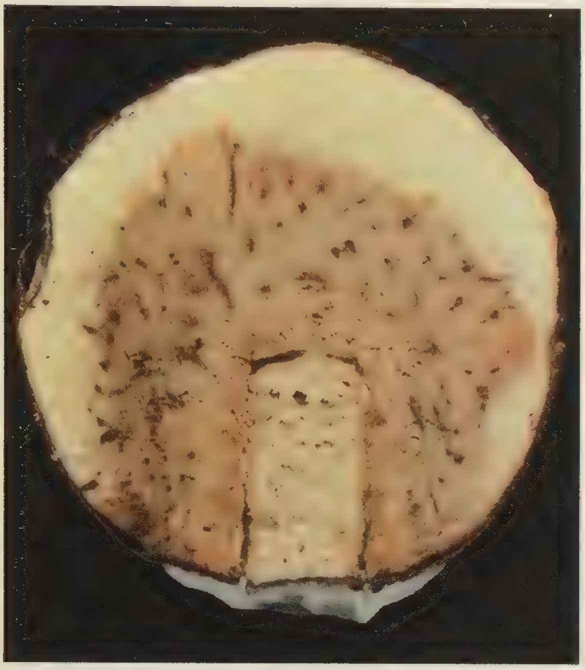
3 HARTER L.L. (1916) Storage rots of economic aroids. *Journal of Agricultural Research* **6**, 549–572 illus.

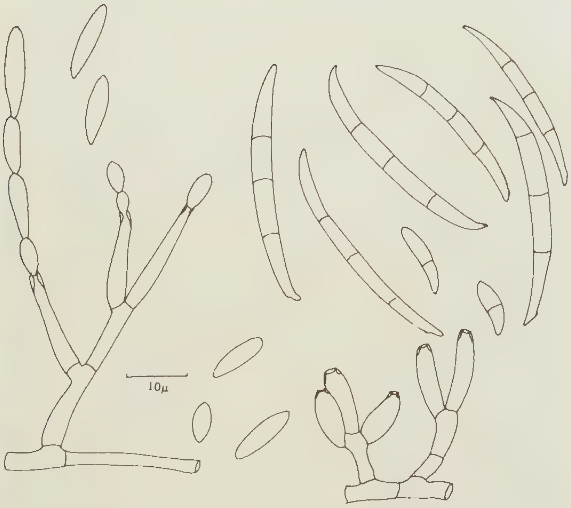
4 SOUZA T.F. d' & MONIZ L. (1968) Root-rot of *Colocasia antiquorum* Schott caused by *Botryodiplodia theobromae* Pat. in Maharashtra state. *Indian Journal of Microbiology* **8**, 45–46.

5 THANKAPPAN M. & MALATHI V.G. (1984) Diseases of aroids. *Indian Farming* **33**(12), 47–49.

FUSARIUM ROT of cocoyams (taros and tannias) caused by *Fusarium* spp.

465





466 *Fusarium moniliforme* (from CMI Descr. No. 22, Booth & Waterston 1964) Conidiophores, microconidia and macroconidia

OCCURRENCE *F. oxysporum* (page 249) has been recorded on taros in the British Solomon Islands (1), while *F. solani* (page 369) occurs on taros in the southern USA, Egypt (2,3) and India (5), and on tannias in Nigeria (4). Nigerian tannias may also be attacked by *F. moniliforme* Sheldon, the conidial state of *Gibberella fujikuroi* (Saw.) Ito.

SYMPTOMS *F. oxysporum* causes a whitish-grey spongy soft rot with a distinct brown margin. Decay usually begins at the base or side of the corm, which may be partially or completely destroyed (1). *F. solani* causes a dry corky or powdery rot with a definite margin. White mould may develop on the surface (5).

BIOLOGY *F. moniliforme* can persist in the soil as minute resting bodies (sclerotia) or in the sexual state (perithecia), while *F. oxysporum* and *F. solani* survive as thick-walled resting spores (chlamydospores). All three species produce abundant asexual spores (conidia) which can cause infection of corms, usually via wounds (5). *F. oxysporum* may induce an evil-smelling soft rot even before harvest (1). All three species are capable of attacking the corms via wounds sustained during harvesting and handling.

CONTROL The chief means of control are careful handling and curing, since fungicide treatment may be ineffective (1). Cool storage is beneficial.

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1 GOLLIFER D.E. & BROWN J.F. (1973) Corm rot of *Colocasia esculenta* caused by *Fusarium oxysporum*. *Plant Disease Reporter* **57,** 701–703 illus.

2 MICHAIL S.H., ELAROSI E. & KHAIRY A. (1969) Dry rot of *Colocasia antiquorum* in U.A.R. (Egypt). *Journal of Phytopathology, U.A.R.* **1,** 23–26 illus.

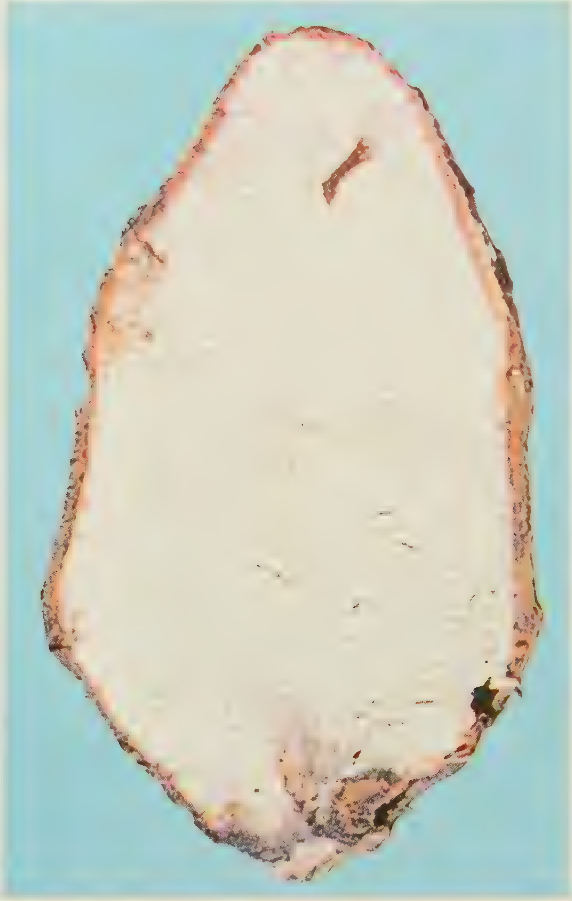
3 MICHAIL S.H. & SALAM M.A. (1981) Studies on dry rot of *Colocasia antiquorum*. *Phytopathologische Zeitschrift* **100,** 157–161.

4 OGUNDANA S.K. (1976) Microbial rot of cocoyam (*Xanthosoma sagittifolium* (L.) Schott) in storage in Nigeria. In *Proceedings of the 3rd International Biodegradation Symposium* (Ed. by J.M. Sharpley & A.M. Kaplan), pp. 645–649 illus. London: Applied Science Publishers Ltd.

5 SHARMA K.D. & UPADHYAY S.K. (1977) Dry rot of *Colocasia esculenta* var. *antiquorum*. *Indian Phytopathology* **30,** 419–420.

PYTHIUM ROT of cocoyams (taros and tannias) caused by ***Pythium*** **spp.**

467



OCCURRENCE Records on taros include *P. deliense, P. myriotylum* and *P. splendens* in the South Pacific Islands (2,3), *P. myriotylum* in Hawaii (4) and Japan, and *P. ultimum* (page 328) in Puerto Rico (1). Wetland taro is especially susceptible, and losses may be catastrophic. Tannias are attacked by *P. myriotylum* in Nigeria and Cameroon (5) and by *P. ultimum* in Puerto Rico (1). In the Caribbean islands pythium rot has been reported on both tannias and taros.

SYMPTOMS Infected tissue is pale and soft, with a crumbly or cheesy consistency (1,2). The lesion is bounded by an irregular but sharply defined margin, beyond which is a wide, pale brown zone of firm undecayed tissue (2). Abundant asexual spores (sporangia) may be produced within the diseased flesh (1). In advanced stages the decay is accompanied by an unpleasant sour odour.

BIOLOGY *Pythium* species are common soil inhabitants, and thrive in warm wet conditions (1). Infection can occur during the growing season, via the roots, which become slimy and rotten (1). The entire plant may collapse, or it may produce only small unmarketable corms (5). Alternatively, normal healthy corms may become infected through wounds sustained during harvesting and handling (1,2). Serious decay can ensue within a few days of harvest (2).

CONTROL It may be feasible to treat the soil with a fungicide (4) and to use only healthy planting material (1,5). Beneficial cultural practices include crop rotation, improved drainage, and avoidance of dense stands. Diseased plants should be promptly removed. Harvested corms can be stored in soil pits to encourage rapid wound healing (2). Corms intended for export should be carefully selected, since post-harvest fungicide treatment is generally ineffective against pythium rot (2). Cool storage can check its development, but decay resumes on return to ordinary temperatures (1). Cultivars differ in their susceptibility to pythium rot (1) and work is in progress to breed more resistant cultivars.

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1 ALVAREZ-GARCÍA L.A. & CORTÉS-MONLLOR A. (1971) Currutaca: a *Pythium* soft rot of *Xanthosoma* and *Colocasia* spp. in Puerto Rico. *Journal of Agriculture of the University of Puerto Rico* **55,** 78–84 illus.

2 JACKSON G.V.H. & GOLLIFER D.E. (1975) Storage rots of taro (*Colocasia esculenta*) in the British Solomon Islands. *Annals of Applied Biology* **80,** 217–230 illus.

3 KERZ-MOEHLENDICK F.H., FLIEGE F.H. & SCHLOESSER E. (1983) Morphology and pathogenicity of four *Pythium* species isolated with root and corm rot of taro, *Colocasia esculenta* (L.) Schott in Western Samoa. *Alafua Agricultural Bulletin* **8,** 53–62 illus.

4 OOKA J.J. & UCHIDA J.Y. (1985) Taro root and corm rot caused by *Pythium myriotylum*. *Phytopathology* **75,** 1316.

5 STEINER K.G. (1981) A root rot of macabo (*Xanthosoma* sp.) in Cameroun, associated with *Pythium myriotylum. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **88,** 608–613.

SCLEROTIUM ROT of cocoyams (taros and tannias) caused by *Corticium rolfsii* Curzi

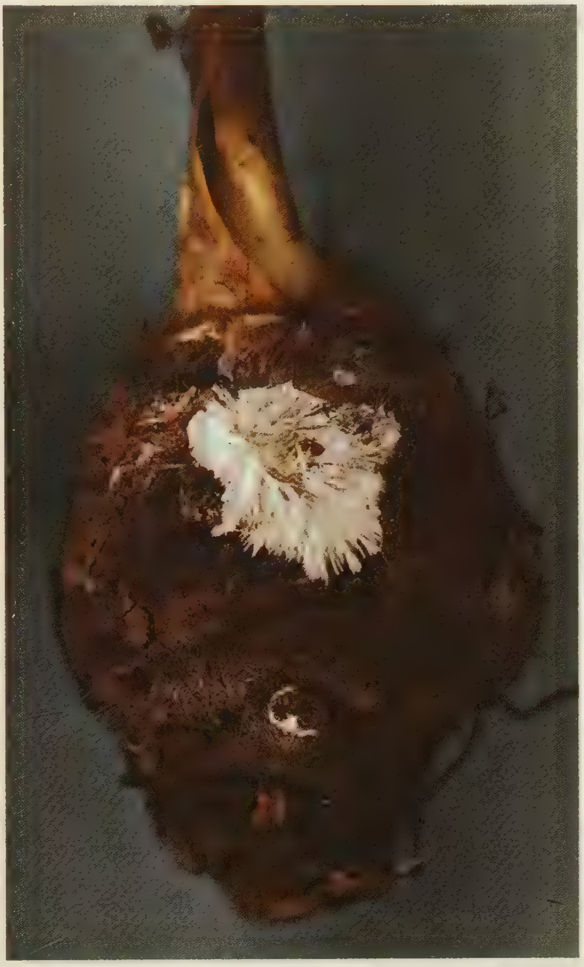
Sclerotial state: *Sclerotium rolfsii* Sacc.

OCCURRENCE This disease has been reported on both taros and tannias in Nigeria (3). Dryland taro is more susceptible than is wetland taro, and records include India (1,2,5), the Philippines, the South Pacific Islands, the southern USA and Hawaii (4).

SYMPTOMS Rotted tissue is pale brown to pinkish, soft but not watery, with a tendency to stringiness. The lesion has a sharply defined margin (4). In humid conditions there is copious development of white mould, which eventually gives rise to small (1–2 mm) spherical resting bodies (sclerotia), white at first, turning to brown (page 255) (3).

BIOLOGY The fungus persists in the soil as sclerotia or mould strands (4); occasionally it may produce the sexual state (a hymenium giving rise to basidiospores) (2). Infection usually occurs at or near the soil surface, since the fungus requires good aeration; this is probably why the disease is uncommon in wetland taro (4). Plants under stress (such as drought) are predisposed to infection (4). Both roots and corms may be attacked, leading to stunting of the plant (4,5). Harvested corms rot in store, and the fungus may be transmitted to new areas in infected planting material (1).

CONTROL It is difficult to eradicate this fungus from infested soil, owing to the longevity of sclerotia and the wide range of susceptible host plants (4). However, partial control has been achieved by deep ploughing (4) or by the practice of intercropping (with plantains). Flooding the field during the early stages of disease development is also effective; infected plants, together with surrounding soil, should first be removed and burned. The crop should be harvested as soon as the corms are mature (4). Cool storage is effective in arresting development of sclerotium rot (3). Alternatively, the corms can be treated with a fungicide; this is also beneficial when they are to be used as planting material (1).



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OTHER DISEASES of cocoyams (taros and tannias)

ASPERGILLUS ROT, caused by *Aspergillus niger* (pages 242, 384), has been recorded on tannias imported into the UK from West Africa. Infected tissue is greyish (**460**) and decay can be extensive at tropical temperatures.

BACTERIAL SOFT ROT, caused by *Erwinia carotovora* ssp. *carotovora* (page 271) or *E. chrysanthemi* (page 302), has been recorded on taros and tannias from Puerto Rico (1) and on taros in the southern USA (3) and the South Pacific Islands (4). These bacteria are widespread and are probably often present with other species of bacteria and fungi, entering via wounds and inducing a soft slimy rot with a repellent odour. If bacterial soft rot is a problem, high humidity storage (in soil pits or in polyethylene bags) should be avoided (4).

BLACK ROT has been recorded on taro in the Pacific Islands, the causal fungus being *Ceratocystis fimbriata* (page 367). Infection is chiefly via wounds, and decayed tissue is soft and dark, with a distinctive odour of bananas.

BLUE MOULD ROT, caused by *Penicillium* spp., is associated with injuries, and tends to be prevalent at temperatures below 20°C.

CYLINDROCARPON ROT can cause important losses of stored taro corms in India (10). The species has been identified as *Cylindrocarpon lichenicola*. It causes maximum damage at 27°C and none at 10°C (10).

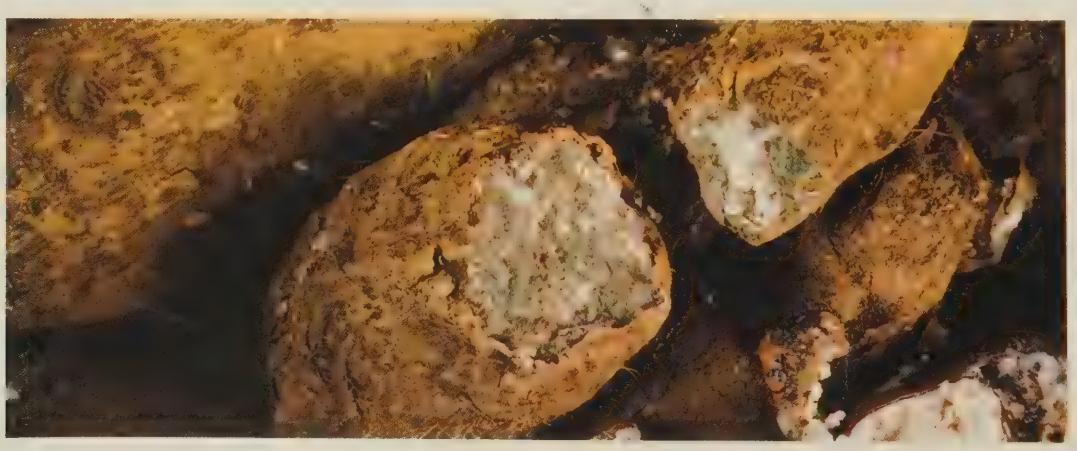
GREY MOULD ROT, caused by *Botrytis* sp., has been found frequently on taros imported into the USA from Puerto Rico (1).

PHYTOPHTHORA ROT of taro corms is one aspect of a major disease caused by *Phytophthora colocasiae* Racib. This fungus is responsible for taro leaf blight, which is common on wetland taro in Hawaii (5) and on dryland taro in the South Pacific Islands (9). Rotting of harvested corms has been reported from the Philippines (2), India (6), Papua New Guinea and the British Solomon Islands (4); lesions are typically pale brown, firm and dry, with a distinct margin, though these characteristics are often altered by the presence of other organisms (4). Phytophthora leaf blight, and hence also corm rot, can be controlled by wide spacing of the plants and by frequent application of a suitable fungicide spray. Dipping corms in a 1% solution of sodium hypochlorite (household bleach) for 2 minutes before storing in polyethylene bags has been shown to give additional protection against *P. colocasiae* when levels of this fungus are particularly high. Work is in progress to breed resistant cultivars (9).

PINK MOULD ROT occurs on taros in India, the causal fungus being *Trichothecium roseum* (page 41).

RHIZOCTONIA ROT has been recorded frequently on both taros and tannias imported into the USA from Puerto Rico (1). *Rhizoctonia solani* (page 307) is one of the causal organisms of the root rot complex of tannias in Nigeria.

RHIZOPUS ROT has been recorded on imported tannias (1), and in India *Rhizopus stolonifer* (page 81) causes a soft rot of stored taros (10). In Hawaii this fungus has caused serious losses in taros stored at moderate temperatures and high humidity while awaiting shipment. The skin of the corm generally remains intact until the rot is very advanced. If the



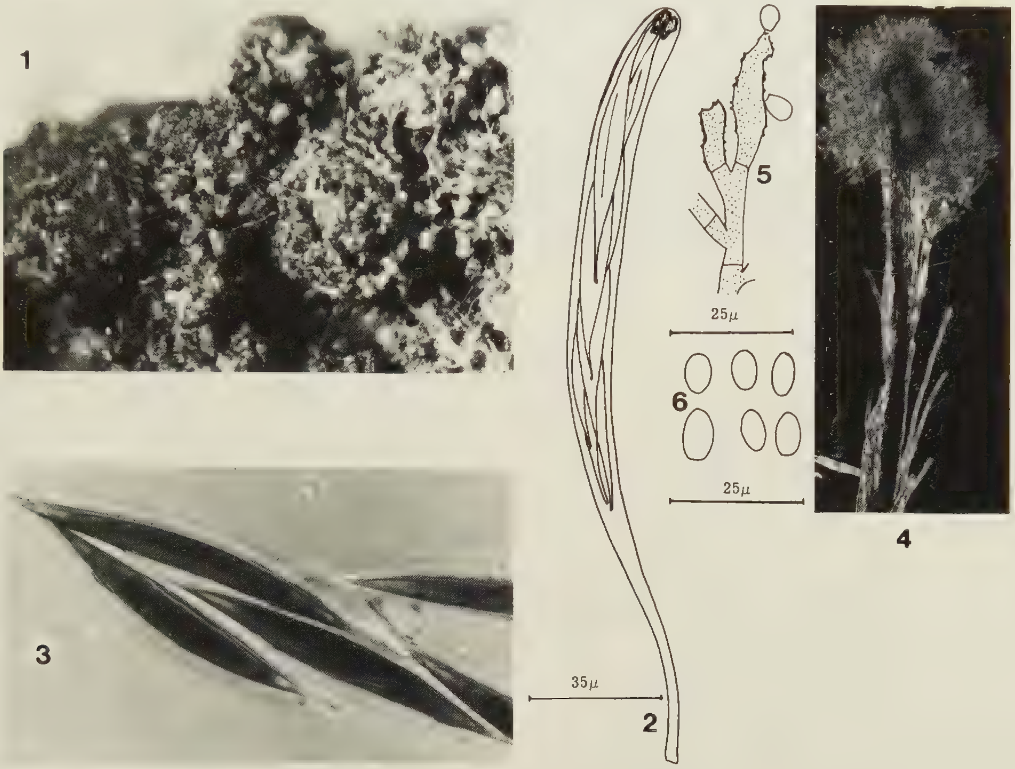
469 Blue mould rot of tannias

skin is broken, a coarse white mould develops, giving rise to characteristic sporeheads (page 372). Decayed tissue is soft and pale, with a cheesy or watery consistency and a slightly yeasty odour.

ROSELLINIA ROT, caused by *Rosellinia bunodes* (Berk. & Br.) Sacc., appears to be more important on taros than on tannias (1).

SOUR ROT of taros has been reported from the southern USA (3) and from India, the causal organism being *Geotrichum candidum* (page 288).

Control of most of these diseases is probably aided by efficient curing of corms after harvest, and the use of a protective chemical dip if subsequent cool storage is unavailable (1).



470 *Rosellinia bunodes* (from CMI Descr. No. 351, Sivanesan & Holliday 1972) 1 perithecia × 13 2 ascus 3 ascospores × 400 4 synnemata × 100 5 conidiogenous branch 6 conidia

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Ginger

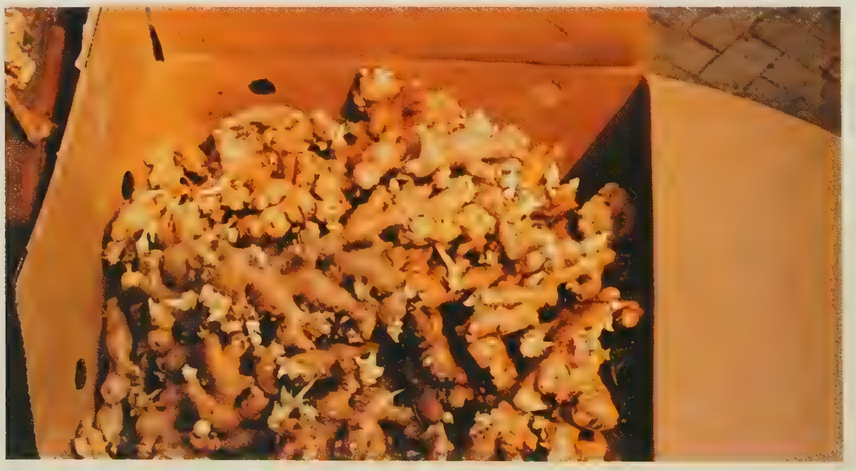
Ginger, *Zingiber officinale* Roscoe, is of very ancient cultivation in Asia. It is a monocotyledonous plant with an erect shoot, long thin leaves and a swollen underground stem or rhizome which is the part used (9). Flowers and seeds are rarely produced, and propagation is exclusively vegetative. Although a perennial plant, ginger is usually grown as an annual crop. Whether fresh or dried, ginger has a distinctive pungency and is generally considered as a spice rather than as an aromatic vegetable (2,3,6). In dried form it figured prominently in early trade, being appreciated in Greece and Rome for its medicinal properties. Living rhizomes were taken to Africa in the 13th century and to central America in the 15th (6).

Production is now widespread in the tropics, and certain countries specialise in the export of particular forms of ginger. From China (via Hong Kong) and Australia comes preserved stem ginger, a sweetmeat made from young tender rhizomes cut into pieces and stewed in syrup (2). Dried ginger is prepared from more pungent mature rhizomes which (whole or split, peeled or unpeeled) are left in the sun or dried artificially until the moisture content is reduced to between 7 and 12% (6). The major exporting countries are India, Jamaica, Nigeria and Sierra Leone, as well as Australia and China. Grinding is usually done in the consumer country, and the spice is used in both sweet and savoury products. Fresh or 'green' ginger is the live rhizome, drying being restricted to a brief (1 or 2 days) curing process intended to facilitate wound healing (1). The fresh ginger of international commerce comes from the West Indies, South and Central America, Hawaii and Mauritius, as well as from several countries in Australasia and the Far East, where ginger is traded extensively. The fresh product is an essential ingredient of many oriental dishes, and commands a high price in international markets.

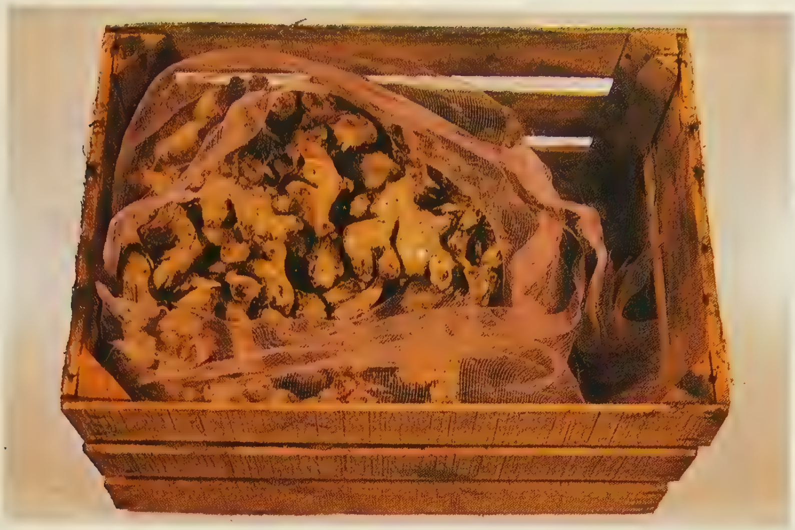
After the processes of washing, fungicide treatment and curing, harvested rhizomes may be kept for several months, depending on the storage facilities available. At temperatures above 20°C weight loss is rapid, through evaporation from cut surfaces and also through respiratory losses, especially after the development of sprouts (5). Waxing can have a perverse effect, sometimes leading to increased moisture loss as a result of stimulation of sprout growth (5). Methods of inhibiting sprouting include irradiation, which may be feasible in some contexts (5). Alternatively, a growth-regulating chemical can be applied; some success is reported for post-harvest treatment (5).

Optimal conditions for ginger storage are a temperature of 13°C and a relative humidity of about 65%; if such an environment can be provided, then a 6-month storage period is possible before weight-loss becomes excessive (1). At temperatures below 13°C ginger is subject to chilling injury, manifest as shrivelling and an increased susceptibility to decay (1). The severity of damage is determined by duration of exposure as well as by actual temperature, and may not become fully apparent until after the ginger has been returned to a warm environment. Symptoms of chilling injury, indistinguishable from those produced by post-harvest mismanagement, can also be the result of pre-harvest frost.

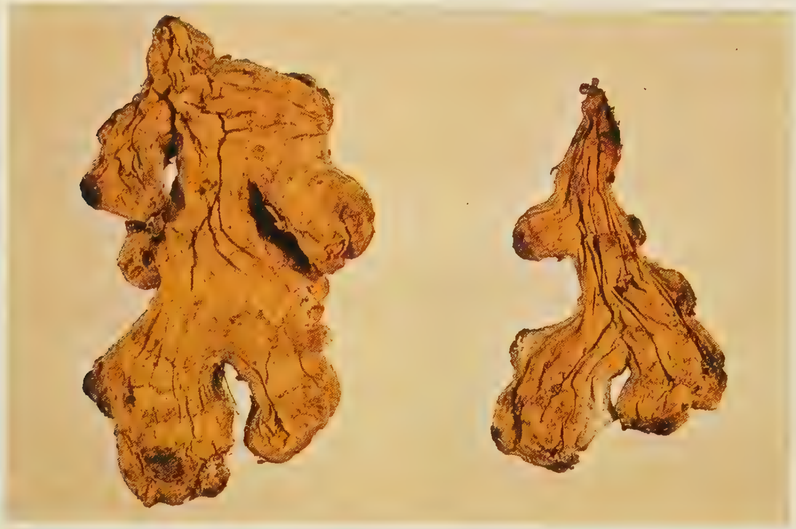
When cool-stored ginger is to be shipped, it should first be air-dried in a warm shaded place (to remove moisture of condensation), and then packed in crates or cartons (1). Shipment is not easy since, in practice, both in ventilated and in refrigerated space, the humidity is inevitably higher than optimal. Consequently, unless a post-harvest fungicide (4,5) has been used, there is a risk of mould growth and decay (7,8,10). Clearly it is advantageous to ensure speedy transportation.



471 Sprouting ginger



472 Chilling injury



473 Close-up of above, showing shrivelling and decay

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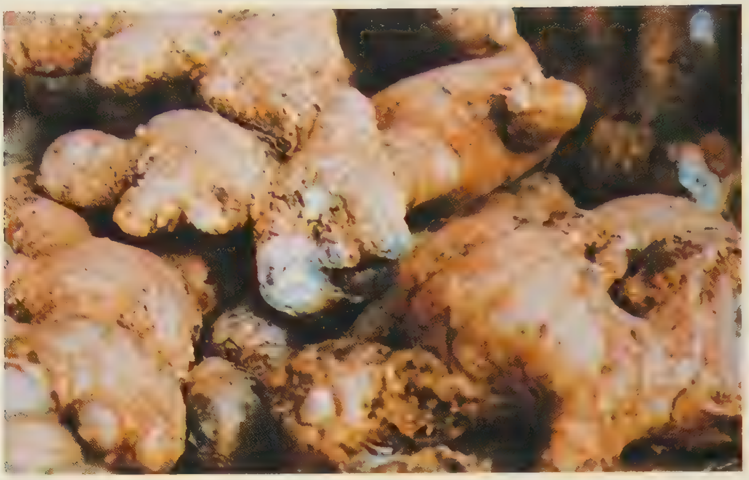
FUSARIUM ROT of ginger caused by
Fusarium **spp.**

474



OCCURRENCE Serious losses have been reported from India (1,2,3), Australia (4) and Hawaii (5). The author has observed fusarium rot in ginger imported into the UK from Brazil, from Nicaragua and from Taiwan. Among the species identified are *F. equiseti* (page 35) (1), *F. oxysporum* f. sp. *zingiberi* (4,5) and *F. solani* (page 369) (2).

SYMPTOMS Early symptoms, visible if an infected rhizome is cut open, include a pale brown discoloration of the vascular strands (5). In contrast to the symptoms produced in bacterial wilt disease (q.v.) there is no milky exudate. The fungus subsequently invades the entire rhizome, which becomes brown, dry and shrivelled (1). If stored in a humid atmosphere, rhizomes may develop a surface growth of white mould.

BIOLOGY These fungi survive in crop debris, and may persist in the soil for many years. Under suitable conditions they produce abundant asexual spores (conidia), and infection of rhizomes occurs by way of growth cracks or through injuries caused by insects, eelworms or cultivating implements. The fungus invades the plant's vascular system and, by preventing water uptake, induces a slow wilt followed by yellowing of the leaves (5). Yields are much reduced and, if infected rhizomes are stored, a dry rot may ensue (1). The organism is transmitted to new areas in infected seed pieces, which either fail to germinate or else give rise to diseased plants (4,5).

CONTROL It may be feasible to disinfect the soil by fumigation or with a fungicide drench. Control of eelworms has been shown to reduce the incidence of fusarium rot. Rhizomes which are to be used for propagation should be carefully selected so as to exclude those which are damaged in any way. Heat treatment of seed-pieces is ineffective (5), but they can be protected by means of a fungicide dip applied before planting (1,4). A fungicide may also be used after harvest to prevent rotting of rhizomes in store (3). Biological control is a possibility, involving a preparation containing an organism which is antagonistic to species of *Fusarium* but is otherwise harmless (1). Efficient curing of rhizomes, followed by storage at moderate temperature, helps to minimise post-harvest losses.

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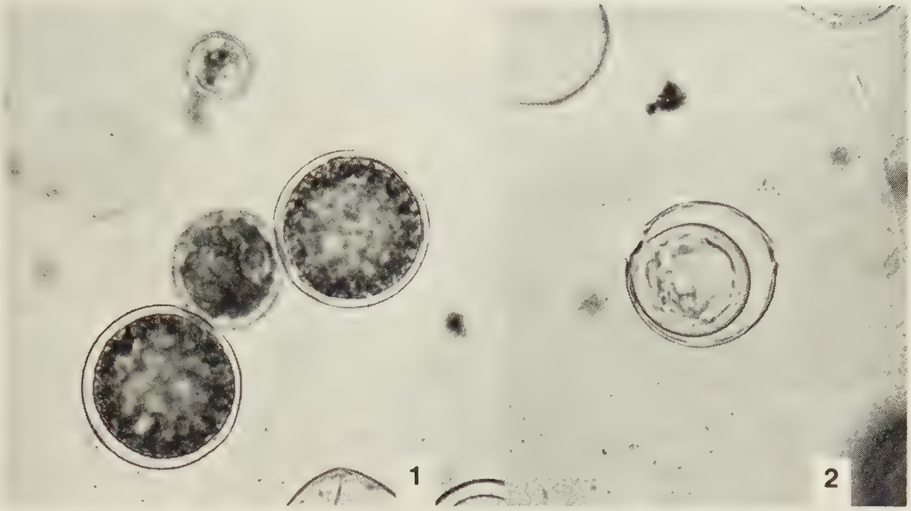
PYTHIUM ROT of ginger caused by *Pythium* spp.



475 *Pythium splendens* (from CMI Descr. No. 120, Waterhouse & Waterston 1966)
1 vegetative hyphal bodies × 500 2 oogonium with antheridia and oospore × 500

OCCURRENCE The disease is widespread in India (1,3,5) and also occurs in Japan (2) and Taiwan (4). Species identified include *P. aphanidermatum* (page 31) (3,5), *P. deliense* (1), *P. myriotylum* (page 117) (4) and *P. zingiberum* (2).

SYMPTOMS Infected rhizomes are discoloured, and become soft as the internal tissue is broken down into a watery mass still enclosed in the tough rind (1,3). Spores (oospores) may (1) or may not (4) be present in damaged tissue.

BIOLOGY The fungi are common in soil (2), surviving unfavourable periods as resting spores (chlamydospores) or in the sexual state (thick-walled oospores) (1,4). Under wet conditions they produce the asexual stage (sporangia), and may also give rise to swimming spores (zoospores). Infection of roots and stem-bases results in 'damping off' or a yellowing and wilting of mature plants (1,4). Heavy rain and temperatures around 30°C favour disease development (4). If the plants survive they may produce infected rhizomes which later rot in store (1,5) or else give rise to diseased shoots.

CONTROL Crop rotation (2) is of limited value because of the longevity of the fungi and their wide host range. The site must be well-drained (1) and, if possible, the soil should be treated with a suitable fungicide. Planting material should be carefully selected and dipped in a fungicide; choice of chemical and time of immersion are critical if germination of the rhizomes is not to be impaired (5). Harvested ginger should be promptly cured, and stored in a cool well-ventilated place.

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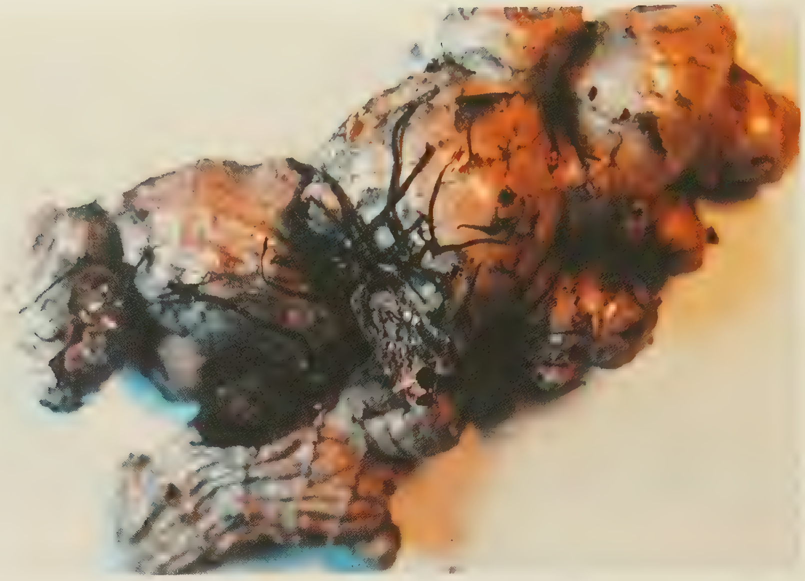
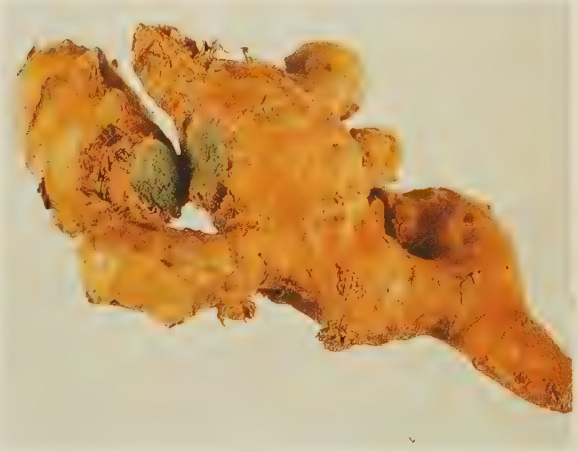
OTHER DISEASES of ginger



476 Armillaria rot

ARMILLARIA ROT is characterised by the development of tough, dark, string-like growths which adhere to the rhizomes. These structures are aggregations of fungal strands (rhizomorphs) of *Armillaria mellea* (Vahl:Fr.) Kummer, commonly known, therefore, as the 'shoe-string fungus'. It is primarily a parasite of tree roots, but can be a problem in ginger grown on recently cleared forest land. Control therefore involves the uprooting of all remaining tree stumps (2).

BACTERIAL SOFT ROT has been responsible for serious losses of stored ginger in Australia (2). The causal organism is *Erwinia carotovora* ssp. *carotovora* (page 271), which is common in soil and may attack rhizomes in poorly drained fields. Infection may also occur during harvest, handling and storage, resulting in a soft wet rot with an offensive odour. Control measures include harvesting at full maturity, careful handling, and efficient curing. Ginger should not be stored in bags which have previously been used for potatoes, carrots, onions, cabbages or cauliflowers, and the storage environment should be cool, dry and well-ventilated (2).



477 Blue mould rot

BACTERIAL WILT DISEASE is caused by *Pseudomonas solanacearum* (page 310) and has been recorded in Australia (2), the Philippines (10), Malaysia, Mauritius and Hawaii. Diseased rhizomes are usually darker than healthy ones; affected flesh is water-soaked and brown, and bacteria ooze from the vascular tissues in milky drops (2,10). The organism is transmitted on seed rhizomes, crop residues, farm implements or machinery, and hygienic practices therefore constitute an important control measure (2). Rotation with non-susceptible crops is beneficial (page 311).

BLACK ROT has been recorded during storage and marketing of ginger in India (1,6,8). The causal fungus, *Memnoniella echinata* (Rev.) Gall., invades injured surfaces, producing a dark mould growth bearing abundant black spores (6). Infected rhizomes become dry and shrivelled.

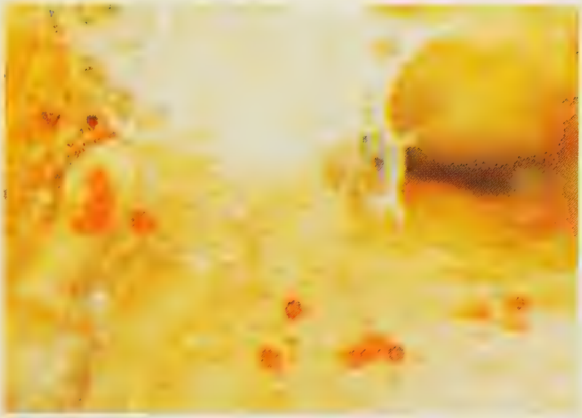
BLUE MOULD ROT is caused by *Penicillium* spp. which are common soil inhabitants. These fungi are capable of colonising injured tissue.

BOTRYODIPLODIA ROT of stored ginger has been reported from India (9). The causal fungus is *Botryodiplodia theobromae* (page 371), and infection results in a dry, brown, powdery rot. It is considered to be a minor disease which is best controlled by careful handling so as to minimise injury and thereby prevent infection.

CHARCOAL ROT, recorded in India, may affect mature rhizomes before and after harvest (4). Symptoms include drying, shrinkage, discoloration and (the diagnostic feature) an abundance of minute powdery black resting bodies (microsclerotia) in the internal tissues, the fungus being *Macrophomina phaseolina* (page 25). Plants are attacked in the field, and infection is thought to take place via injuries made by weevil grubs. Above-ground symptoms range from slight yellowing to severe blight. Control measures should be directed against the insect pests.

GREY ROT, caused by *Trichurus spiralis* Hasselbr., has been reported from India (6,8). The fungus invades rhizomes by way of injuries or cut ends and incites a dry rot. A dense mould develops, characterised by minute greyish-black structures (synnemata) bearing spores (conidia).

RED ROT may be caused by *Nectria inventa* Pethybr., which occurs in India (1,6). Small, irregular, shiny, light brown patches enlarge and coalesce, becoming brick-red with age. The skin is easily detached, and the rhizome dries and shrinks. Only the asexual state of the fungus was observed, viz. *Verticillium luteo-album*. In the Philippines red rot is caused by *Nectriella zingiberi* Stevens & Atienza. Affected tissue is dry, and rarely deeper than 3 or 4 mm. The superficial red colour is provided by dense clumps of minute brightly coloured bodies (perithecia), the sexual stage of the fungus, of which a colourless variant also exists (7).


478 Red rot, showing perithecia

ROSELLINIA ROT, reported from the Philippines, is said to be common in rhizomes which are left for a time on the ground after harvest (7). A white mould infects the tissue, which becomes dark, especially in the vascular region, and dries out leaving a cavity. Severely rotted tissue may support globose bodies (perithecia) up to 2 mm across, the causal organism being *Rosellinia bunodes* (page 357).

SCLEROTIUM ROT *Sclerotium rolfsii* (page 255) is important in India (1) and has also been recorded in Sri Lanka and Australia (2). Rhizomes may appear to be superficially contaminated with white mould, or there may be substantial rotting and shrinkage (1,3,5). Small (1–2 mm) spherical resting bodies (sclerotia) develop on the mould; they are at first white and later brown. These sclerotia can survive for years in the soil.

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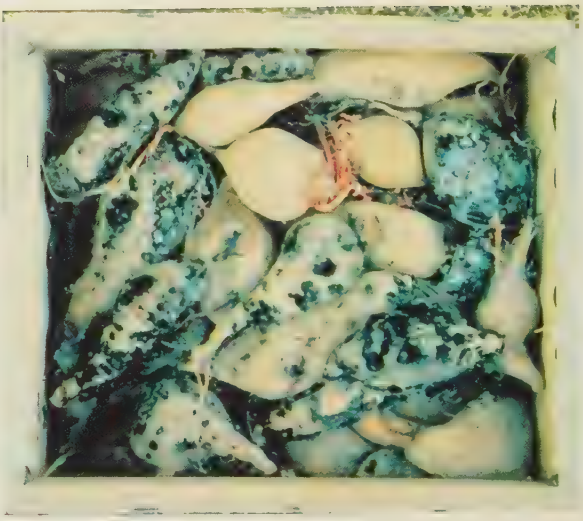
Sweetpotatoes

The **sweetpotato**, *Ipomoea batatas* (L.) Lam., had its origins in tropical America, where it has been grown for several millennia. The crop is also of ancient cultivation in Polynesia, though there is some uncertainty as to how it came there. To the rest of the world the sweetpotato remained unknown until the voyages of discovery in the 15th and 16th centuries, when Spanish and Portuguese travellers introduced it into Europe, Africa and the Far East. There was much early confusion with what is now called the potato (page 296). Current world production of sweetpotatoes is of the order of 130 million tonnes, of which more than four fifths is grown in China. Other producers include Uganda, Indonesia, Viet Nam, Japan, India, the Philippines, Brazil, Rwanda and South Korea. In some countries, for example the USA and Japan, the fresh product is often processed by canning, freezing or chipping; other uses of sweetpotato are as an animal feedstuff and as a basis for the industrial production of starch, syrup and alcohol (2,9).

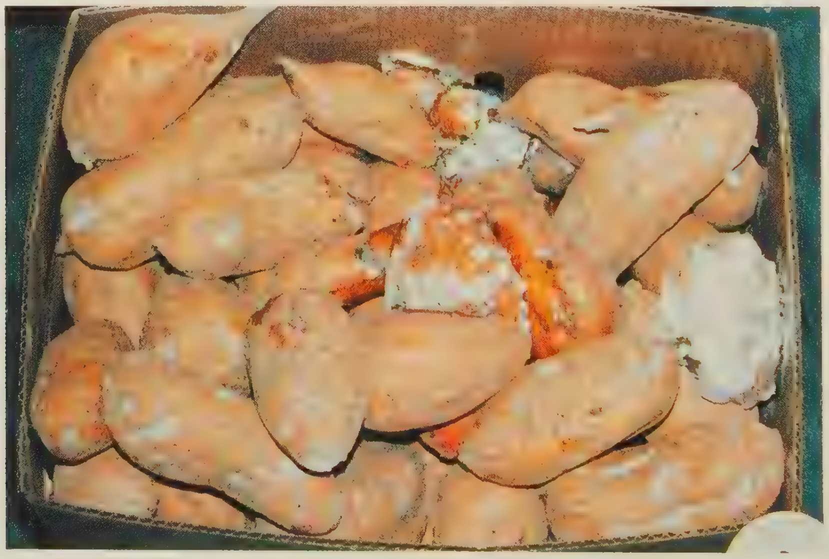
The plant (a dicotyledon) belongs to the family Convolvulaceae and possesses rampant, vine-like stems (8,9). Certain of its roots undergo a complex thickening, becoming swollen and tuber-like. Shape, size, colour and texture are highly variable, but two main types are recognised on the basis of the characteristics of the cooked flesh; 'dry types' have firm white or yellow flesh which is only mildly sweet, while 'moist types' possess soft orange flesh with a substantial sugar content (8). In the USA the latter are sometimes referred to as 'yams', despite being quite unrelated to the true yams (q.v.). This ambiguous usage can cause misunderstandings between importers and exporters in different parts of the world.



479 Java black rot

Most sweetpotatoes are grown for local consumption. In the tropics roots can be harvested over an extended period and storage is rarely required (7); propagation is usually by means of stem-cuttings (8). In cooler regions harvested roots are stored through the winter, and some must be set aside for use as planting material in the following season.

For successful storage and marketing it is necessary to subject the harvested roots to a preliminary process of curing, which has several beneficial effects (6,10). It causes biochemical changes which later enhance palatability and sweetness in the cooked product. It also results in toughening of the skin and healing of wounds, thereby reducing the risk of post-harvest infection and decay. Curing



480 Fusarium rot

should precede any fumigation treatment (against insects), which may be a necessary quarantine procedure. The curing process should take place in the store, to obviate the need for further handling which would cause fresh wounds. Optimal conditions are a temperature approaching 30°C and a relative humidity of 90 to 95%; in such an environment curing is accomplished within a few days (6,8,10). Good ventilation is essential, during both curing and subsequent storage. Under warm ambient conditions healthy sweetpotatoes may be stored for a few weeks. A sprout suppressant (applied before or after harvest) is sometimes used, and a post-harvest hot-water dip (7) can extend storage life significantly by controlling microbial decay (1,3,4,8). If refrigeration is available, healthy well-cured roots can be kept for several months (10). The optimal storage temperature, between 13° and 16°C depending on cultivar, is not low enough to prevent the development of established fungal infections. Sub-optimal temperatures, on the other hand, can cause chilling injury (q.v.) (5).

To prepare sweetpotatoes for market, they are first washed to remove the adhering soil, then sorted and graded (2). If the roots have been cool-stored it can be advantageous to warm them in a hot-water bath in order to facilitate a second curing (necessitated by the inevitable wounding caused by renewed handling). Alternatively the roots can be protected by means of a fungicide dip, which may be more effective than re-curing. Methods of packaging include cartons and wooden crates, and shipment may be in ventilated or in refrigerated space (2,6). With regard to compatibility of different commodities, it should be noted that sweetpotatoes can give out significant quantities of ethylene, following wounding or decay.

481 Sprouting

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1 CLARK C.A. & MOYER J.W. (1988) *Compendium of sweet potato diseases.* St Paul, Minnesota: American Phytopathological Society, 74 pp illus.

2 EDMOND J.B. & AMMERMAN G.R. (1971) *Sweet potatoes: production, processing and marketing.* Westport, Conn.: AVI Publishing Co., 334 pp.

3 HARTER L.L. & WEIMER J.H. (1929) A monographic study of sweet potato diseases and their control. *Technical Bulletin of the United States Department of Agriculture* No. 99, 117 pp.

4 LAURITZEN J.I. (1935) Factors affecting infection and decay of sweet potatoes by certain storage rot fungi. *Journal of Agricultural Research* **50,** 285–329 illus.

5 PICHA D. (1987) Chilling injury, respiration, and sugar changes in sweet potatoes stored at low temperature. *Journal of the American Society for Horticultural Science* **112,** 497–502.

6 PLUMBLEY R.A. (1979) Transport and handling of sweet potatoes. *Report of the Tropical Products Institute* No. 864, 19 pp.

7 SCRIVEN F.M., NDUNGURU G.T. & WILLS R.B.H. (1988) Hot water dips for the control of pathological decay in sweet potatoes. *Scientia Horticulturae* 35, 1–5.

8 STEINBAUER C.E. & KUSHMAN L.J. (1971) Sweet potato culture and diseases. *Agriculture Handbook of the United States Department of Agriculture* No. 388, 74 pp illus.

9 VILLAREAL R.L. & GRIGGS T.D. (Eds.) (1982) Sweet potato. *Proceedings of the 1st International Symposium.* Tainan, Taiwan: Asian Vegetable Research and Development Center, 493 pp.

10 WALTER W.M., HAMMETT L.K. & GIESBRECHT F.G. (1989) Wound healing and weight loss of sweet potatoes harvested at several soil temperatures. *Journal of the American Society for Horticultural Science* **114,** 94–100.

See also:

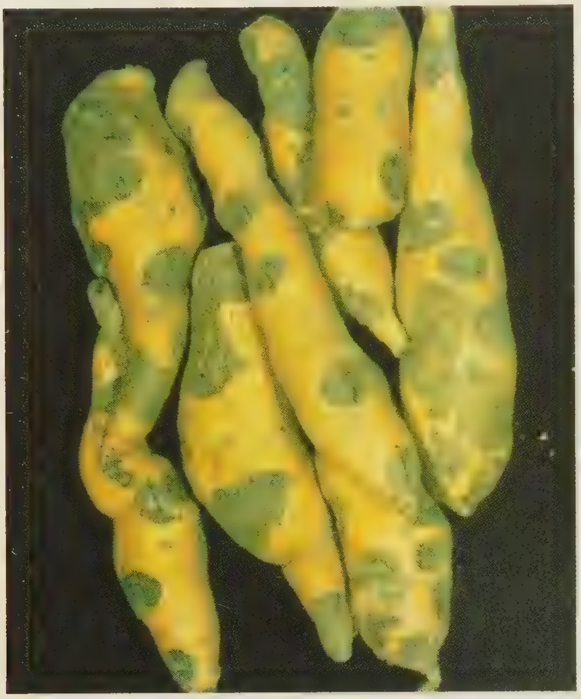
CEPONIS M.J. & BUTTERFIELD J.E. (1974) Retail and consumer losses in sweet potatoes marketed in metropolitan New York. *HortScience* **9,** 393–394.

BLACK ROT of sweetpotatoes caused by

Ceratocystis fimbriata **Ell. & Halst.**

Conidial state: *Thielaviopsis* sp. (syn. *Chalara* sp.)

482



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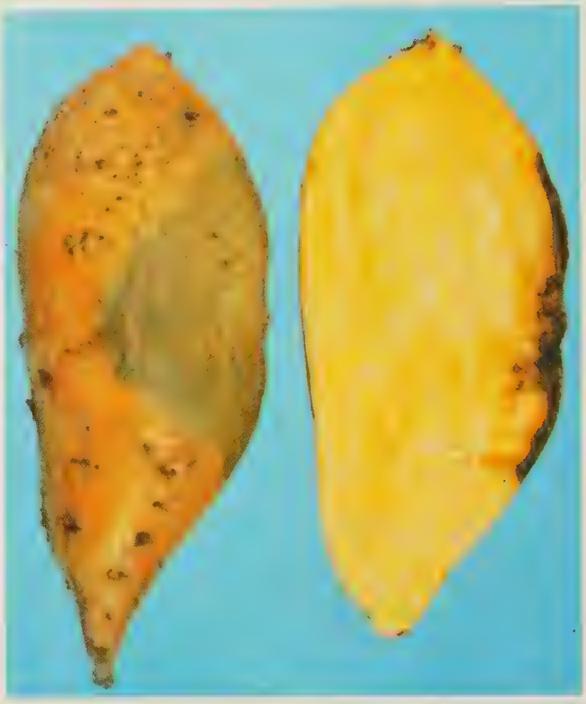
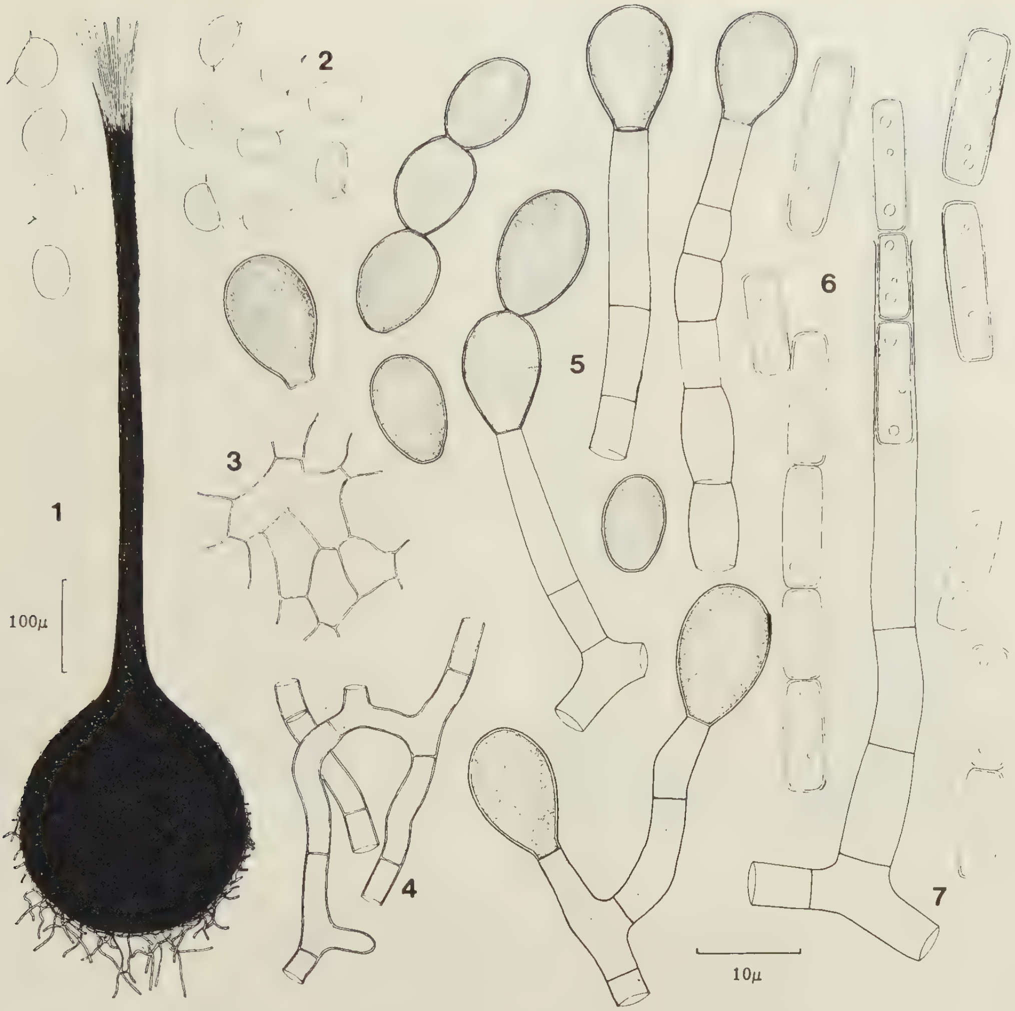


OCCURRENCE Black rot is an important disease; most references are from the USA (1,3,6,7,8,9) but it has also been recorded in other tropical, subtropical and warm temperate countries such as Haiti, Peru, Japan (4), Papua New Guinea and New Zealand (2,10). It was introduced into the Azores on sweetpotatoes imported from the USA, and into China from Japan.

SYMPTOMS The slightly sunken circular lesions are at first brown and later greenish-black (2). Associated with mature lesions are minute black bodies (perithecia) with long necks which appear to the naked eye as dark bristles (9). Even in advanced stages the rot remains firm and generally shallow, but when the root is cooked, the entire flesh tastes bitter (10).

BIOLOGY The fungus persists in soil and crop debris (4), surviving hot dry periods as thick-walled resting spores (chlamydospores) (2). It may also exist in the sexual state (perithecia giving rise to ascospores) (9) or the asexual state (conidia) on infected sweetpotatoes used as seed (3). Young shoots are thereby infected, and subsequently the main stem and the progeny tubers. Spore dispersal is by wind, rain or animals (10). Infection of healthy plants is facilitated by wounds made by insects or rodents (2,4,8). Roots may appear healthy at harvest but later rot in store or during marketing, especially if they have been washed (7,8,10). Development and spread of the disease is rapid at temperatures around 25°C (8), and infected root tissue gives off significant amounts of ethylene gas (5).

CONTROL Crop rotation is beneficial, since most other crops are unaffected by this fungus (2,10). It is necessary to disinfect the seed-bed if a clean site is unavailable (8,10). Contaminated seed roots should be heat-treated for 1 to 3 days or else dipped in a heated fungicide before planting (2,3,10). It may be easier to propagate plants from healthy stem-cuttings (10). Harvested roots should be handled carefully, heat-treated (7) for no more than 24 hours, and cured at approximately 35°C for 2 to 10 days (6). Subsequently, a storage temperature of 10° to 12°C is effective for contaminated roots, especially if the relative humidity is less than 90%; such conditions are unfavourable to infection. If infection has already occurred, however, then decay ensues even at 9° to 10°C and immediate marketing is advisable (8). Alternatively a post-harvest fungicide dip can be applied immediately after harvest. Sweetpotato cultivars differ greatly in their susceptibility to black rot (1).



484 *Ceratocystis fimbriata* (from CMI Descr. No. 141, Morgan-Jones 1967) 1 perithecium 2 ascospores 3 surface of perithecium wall 4 mycelium 5 chlamydospores 6 conidia 7 conidiophore

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1 CHEO P.C. (1953) Varietal differences in susceptibility of sweet potato to black rot fungus. *Phytopathology* **43,** 78–81 illus.

2 CHEVAUGEON J. (1957) *Ceratocystis fimbriata* Ellis & Halstead. *Revue de Mycologie* **22** (*Supplément Colonial 2*), 45–60 illus.

3 DAINES R.H. (1971) The control of black rot of sweet potatoes by the use of fungicide dips at various temperatures. *Phytopathology* **61,** 1145–1146.

4 GOTO K., SUSUKI N., KONDO S. & MIYAJIMA M. (1954) [On the soil infection of blackrot of sweetpotato and its transmission by fieldmouse.] *Bulletin of the Division of Plant Breeding and Culture, Tokai-Kinki Agricultural Experiment Station* No. 1, 138–150 illus.

5 HYODO H. & URITANI I. (1984) Ethylene production in sweet potato root tissue infected by *Ceratocystis fimbriata. Plant and Cell Physiology* **25,** 1147–1152.

6 KUSHMAN L.J. (1959) Curing of Porto Rico sweet potatoes at 95°F for prevention of black rot in storage. *Proceedings of the American Society for Horticultural Science* **73,** 467–472 illus.

7 KUSHMAN L.J. & COOLEY J.S. (1949) Effect of heat on black rot and keeping quality of sweet potatoes. *Journal of Agricultural Research* **78,** 183–190 illus.

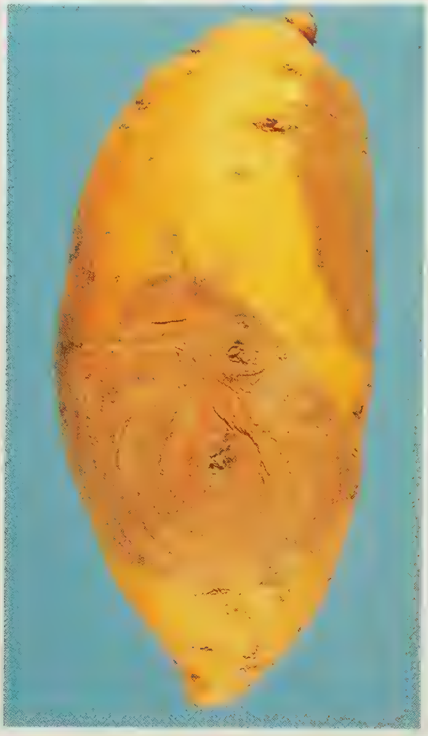
8 LAURITZEN J.I. (1926) Infection and temperature relations of black rot of sweet potatoes in storage. *Journal of Agricultural Research* **33,** 663–676.

9 OLSON E.O. (1949) Genetics of *Ceratostomella.* 1. Strains in *Ceratostomella fimbriata* (Ell. & Hals.) Elliott from sweet potatoes. *Phytopathology* **39,** 548–561 illus.

10 SLADE D.A. (1960) Black rot an important disease of kumaras. *New Zealand Journal of Agriculture* **100,** 375–378 illus.

FUSARIUM ROT of sweetpotatoes caused by ***Fusarium* spp.**

485



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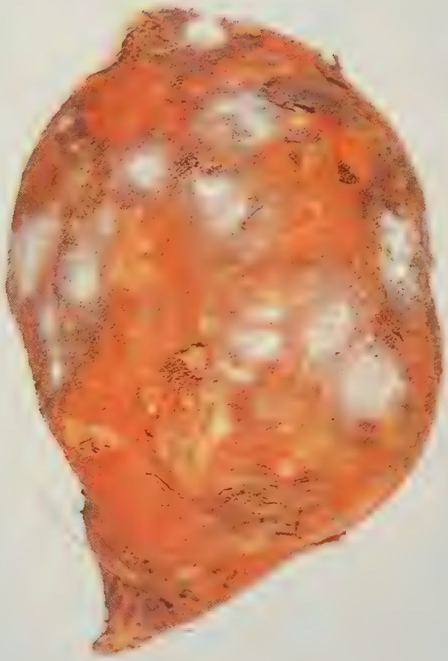

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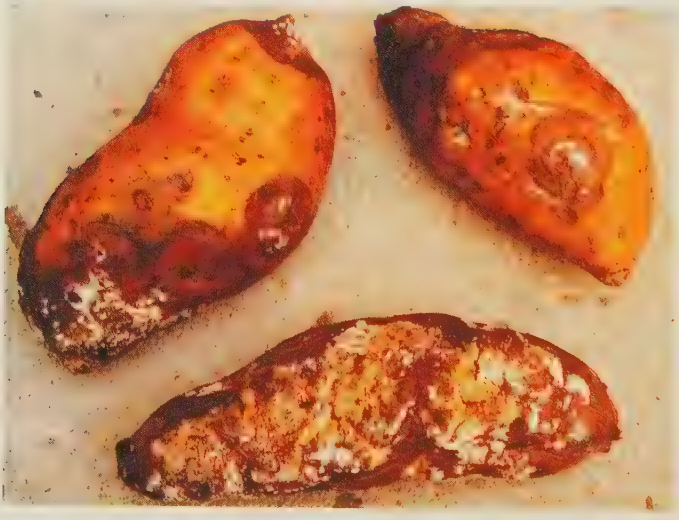
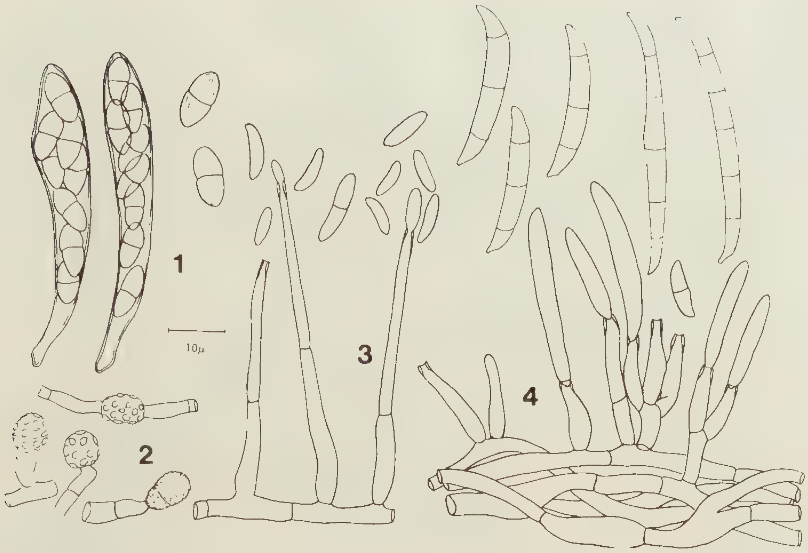


OCCURRENCE *F. oxysporum* (page 249) has been recorded on sweetpotatoes in the USA (4,6), while *F. solani* (Mart.) Sacc. is reported from the USA (1,2,7,9) and China (5). Both fungi are common throughout the world.

SYMPTOMS The type of decay is rather variable. End rot, caused by either species, is characterised by a dry decay, at one or both ends of the fleshy root, the lesions being brown with a dark margin (2). Infected tissue shrivels, sometimes forming cavities filled with white mould. Surface rot, again caused by either species, consists of pale brown circular lesions, often centred on a broken rootlet. Decay remains shallow, but the lesions constitute a disfiguring blemish (4,6). *F. solani* can also cause an invasive rot, starting at wounds and eventually affecting the entire root (7).



488 *Fusarium solani* (from CMI Descr. No. 29, Booth & Waterston 1964)
1 asci and ascospores 2 chlamydospores 3 microconidia and conidiophores 4 macroconidia and conidiophores

BIOLOGY Both fungi persist in the soil as thick-walled resting spores (chlamydospores). They also produce asexual spores (conidia) which are dispersed by wind and water. Infection may occur in the field (4,5), via rootlets or through growth cracks. More commonly, however, roots contaminated with spores are invaded through wounds sustained at harvest (2,7). Wet soil conditions increase the likelihood of infection (4). Post-harvest decay tends to be slow, and symptoms may not become apparent for several weeks (4,7,8). If infected roots are used as seed, the daughter plants may develop stem cankers (3).

CONTROL Soil solarization (page 132) can be effective in reducing the level of soil infestation (9). It may be advisable to use fungicide treatments on planting material and on the growing crop (5). Care should be taken to minimise injury during harvesting and handling, especially if the crop has been lifted from wet soil (4). Curing is best done in store rather than on the field. Alternatively, the roots can be dipped in a fungicide immediately after harvest (7). Some sweetpotato cultivars are resistant to fusarium rot (5), and breeding work continues (3).

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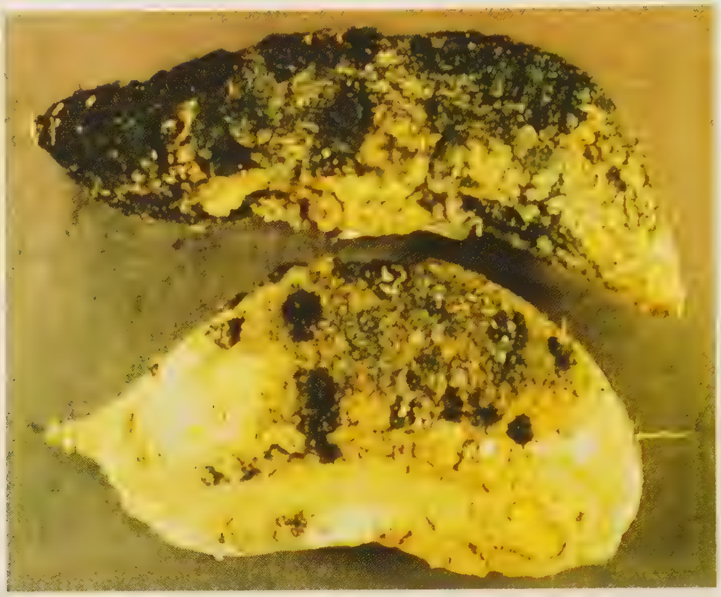
1 CAMPBELL T.M. & COLLINS W.W. (1988) Effects of *Fusarium solani* root rot and presprouting treatments on 'Jewel' sweet potato plant production. *Canadian Journal of Plant Pathology* **10,** 240–245.
2 CLARK C.A. (1980) End rot, surface rot, and stem lesions caused on sweet potato by *Fusarium solani. Phytopathology* **70,** 109–112 illus.
3 CLARK C.A., RANDLE W.M. & PACE C.S. (1986) Reactions of sweet potato selections to *Fusarium* root and stem canker caused by *Fusarium solani. Plant Disease* **70,** 869–871.
4 HARTER L.L. & WEIMER J.L. (1919) The surface rot of sweet potatoes. *Phytopathology,* 465–469 illus.
5 LIU Q., YE D. & XU Z. (1982) [On the root rot of sweet potato.] *Acta Phytopathologica Sinica* **12**(3), 21–28 illus.
6 MARTIN W.J. & PERSON L.H. (1951) Surface rot of Porto Rican sweet potatoes. *Phytopathology* **41,** 228–230 illus.
7 NIELSEN L.W. & MOYER J.W. (1979) A *Fusarium* root rot of sweetpotatoes. *Plant Disease Reporter* **63,** 400–404 illus.
8 SCOTT L.E., KANTZES J. & BOUWKAMP J.C. (1972) Clonal differences in the incidence of surface rot (*Fusarium* spp.) on sweetpotato. *Plant Disease Reporter* **56,** 783–784.
9 STEVENS C., TANG A.Y., KHAN V.A. & WILSON M.A. (1986) Effect of soil solarization on soilborne pathogens of sweet potato. *Phytopathology* **76,** 1068 [Abstract].
10 WOLLENWEBER H.W. (1914) Identification of species of *Fusarium* occurring on the sweet potato, *Ipomoea batatas. Journal of Agricultural Research* **2,** 251–285 illus.

JAVA BLACK ROT of sweetpotatoes caused by ***Botryodiplodia theobromae*** **Pat.**

489



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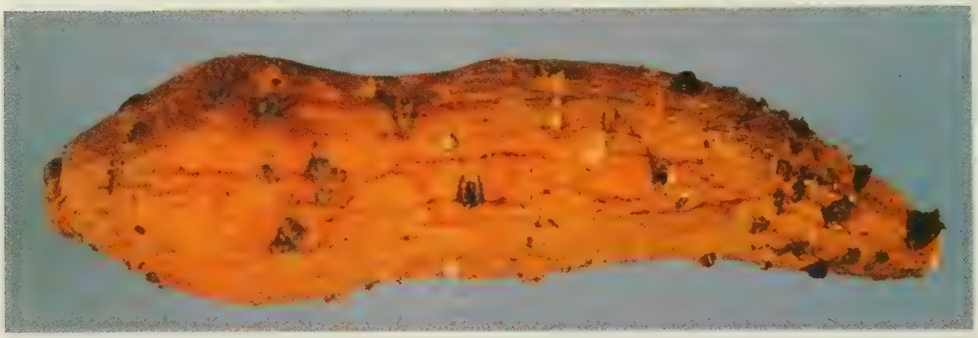
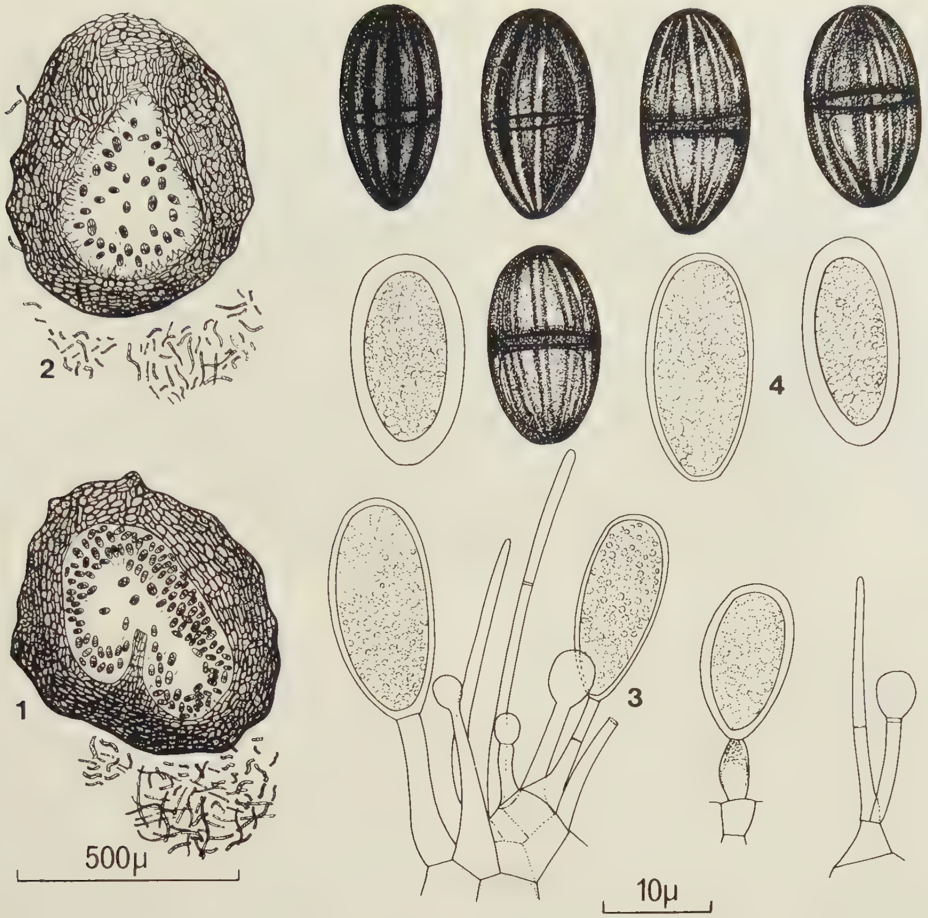


OCCURRENCE The disease was first observed in consignments imported into the USA from Java (5), and the same fungus has been intercepted in sweet potatoes shipped from Egypt to Italy (10). It is especially common in the tropics, for example Nigeria (1,2), Bangladesh (6), the Philippines (4,8) and the southern USA (3,5,7,9).

SYMPTOMS Infected tissue is at first yellowish-brown and fairly firm, later darkening to black. After some weeks affected roots become mummified, and the skin is pimpled with minute black bodies (pycnidia) (5,9,10).

BIOLOGY The fungus is a common soil inhabitant (7), persisting in the asexual state (pycnidia giving rise to conidia). The spores are disseminated by rain, and roots are likely to be contaminated at harvest (7). Infection occurs via wounds sustained during harvesting and handling (6), and the subsequent rate of decay is determined by conditions of storage or transport (10). Temperatures around 30°C favour disease development (3). If contaminated sweetpotatoes are used as seed, the sprouts (shoots) may become infected, and subsequently the progeny roots (3).

CONTROL Seed sweetpotatoes should be dipped in a fungicide before being planted (3). Harvested roots should be promptly washed, dipped in a fungicide, and cured at high humidity to ensure rapid wound healing (7). The cured roots may with advantage be stored at a temperature between 13° and 16°C. Lower temperatures are inadvisable since any consequent chilling injury would predispose the roots to decay on removal to warmer conditions (2). Even under optimal conditions, stored roots become progressively more susceptible to invasion, and there is a risk of new infections taking place during handling for marketing (7). Some sweetpotato cultivars are resistant to the disease (4,8).



491 *Botryodiplodia theobromae* (from CMI Descr. No. 519, Punithalingam 1976)
1,2 pycnidium 3 conidiogenous cells and conidia 4 conidia

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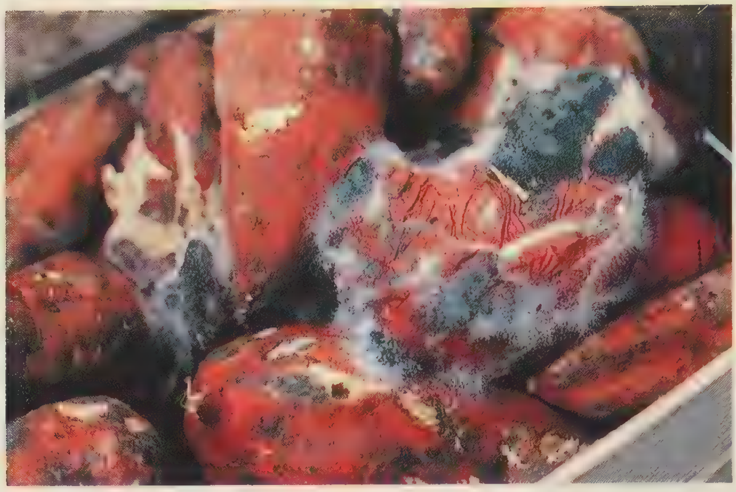
1 ARENE O.B. & NWANKITI A.O. (1978) Sweet potato diseases in Nigeria. *PANS* **24,** 294–305 illus.
2 ARINZE A.E. & SMITH I.M. (1982) Effect of storage conditions on the resistance of sweet potato tissues to rotting by *Botryodiplodia theobromae* and other fungi. *Journal of Stored Products Research* **18,** 37–42.
3 DAINES R.H. (1959) The effect of plant bed temperature and fungicide treatments on the occurrence of Java black rot disease of sweet potato sprouts. *Phytopathology* **49,** 252–254 illus.
4 DALISAY R.F., DIVINAGRACIA G.G. & MENDOZA E.M.T. (1987) Screening sweet potato roots for resistance to Java black rot caused by *Diplodia tubericola* (Ell. & Ev.) Taubenh. *Philippine Journal of Crop Science* **12,** 33–37.
5 HARTER L.L., WEIMER J.L. & ADAMS J.M.R. (1918) Sweet potato storage rots. *Journal of Agricultural Research* **15,** 337–368 illus.
6 JENKINS P.D. (1982) Losses in sweet potatoes (*Ipomoea batatas*) stored under traditional conditions in Bangladesh. *Tropical Science* **24,** 17–28.
7 LO J.-Y. & CLARK C.A. (1988) Sources of inoculum and infection courts of *Diplodia gossypina* on sweet potato. *Phytopathology* **78,** 1442–1446.
8 PALOMAR M.K., SOLIS A.D. & BANDALA H.S. (1980) Sweet potato tuber rot disease in the Philippines. *Annals of Tropical Research* **2,** 111–121.
9 TAUBENHAUS J.J. (1913) The black rots of the sweet potato. *Phytopathology* **3,** 159–166 illus.
10 TROTTER A. (1927) Sulla presenza della '*Lasiodiplodia tubericola*' Ell. et Evr. in Egitto ('Java black rot') e sul pericolo della sua introduzione in Italia. *Bolletino della R. Stazione di Patologia Vegetale (NS)* **7,** 93–98 illus.

RHIZOPUS ROT of sweetpotatoes caused by ***Rhizopus* spp.**

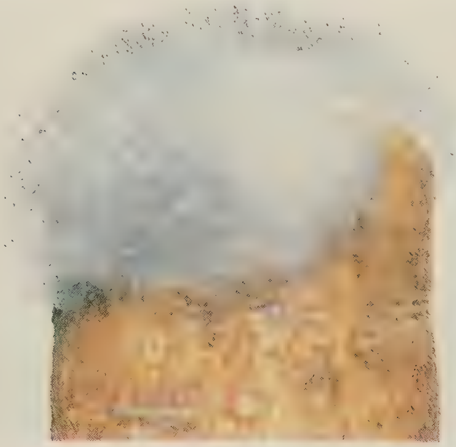
492



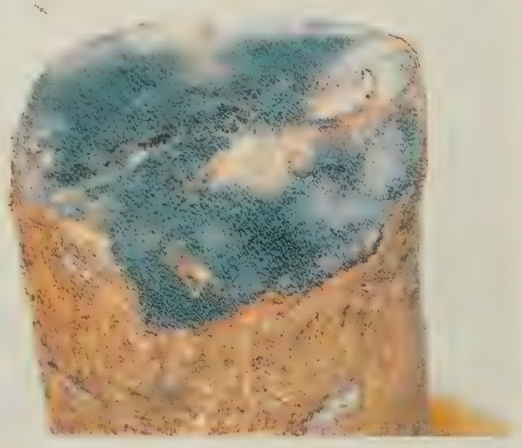
OCCURRENCE The disease is widespread and important, causing heavy losses (6,10). It probably occurs in sweetpotatoes from all producer countries, but most investigations have been carried out in the USA. Numerous species of *Rhizopus* have been recorded (3,4). *R. stolonifer* (Ehrenb. ex Fr.) Lind is by far the most common; *R. oryzae* Went & Prinsen Geerligs and *R. tritici* are among the others studied (4,5).

SYMPTOMS Decay often begins at one end of the root (9). Alternatively it may start on the shoulder, causing a sunken lesion which tends to encircle the root. In a dry atmosphere rotting may be arrested; the flesh remains firm but begins to shrivel. Under humid conditions affected tissue is soft and watery; in places where the skin has ruptured there is copious development of coarse white mould, bearing characteristic globular spore-heads (sporangia) which turn from white to black as they mature (2,10). Colonization of the entire root can occur within a few days, and mould spreads to adjacent roots, causing 'nests' of decay (10).

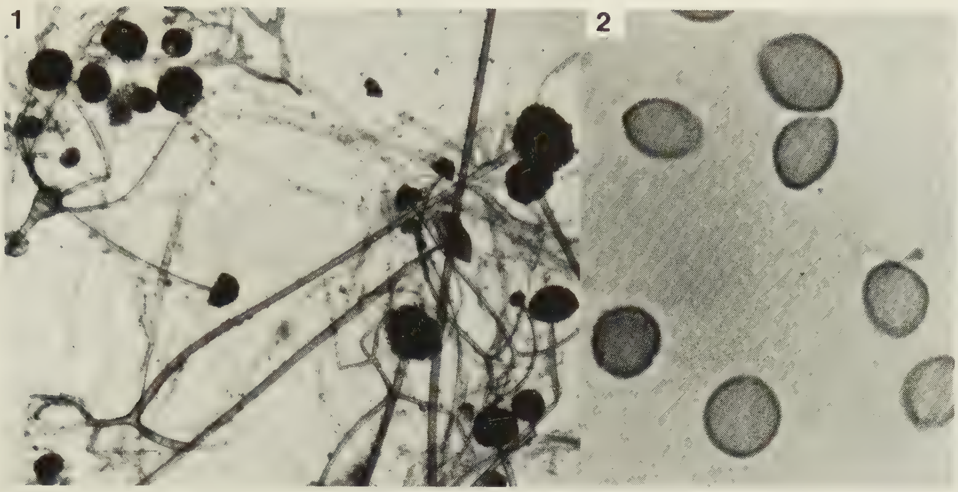
BIOLOGY Species of *Rhizopus* are common in soil and in the atmosphere, and harvested sweetpotato roots are likely to be contaminated with spores. Wounds predispose the roots to attack, and the root-tip is especially susceptible to invasion because the natural presence of dead tissue is advantageous to the fungus (9). Once established, it is capable of attacking healthy uninjured tissue. Infection and decay are greatly influenced by environmental conditions during transport and storage. Infection is especially likely if the relative humidity is between 75 and 85%; a drier atmosphere inhibits the mould, while a more humid atmosphere promotes wound-healing which protects the root from attack (5). If,



493 *Rhizopus stolonifer*



494 *Rhizopus oryzae*



495 *Rhizopus oryzae* (from CMI Descr. No. 525, Lunn 1977) 1 sporangiophores, rhizoids, columellae and sporangia × 100 2 sporangiospores × 2500

however, uncured roots are first allowed to wilt and then placed in a humid environment, the mould may be able to invade before the roots can develop a defence, and catastrophic rotting usually follows (5). The optimal temperature for decay is approximately 20°C for *R. stolonifer* and in the region of 30°C for *R. oryzae* and *R. tritici* (3,4).

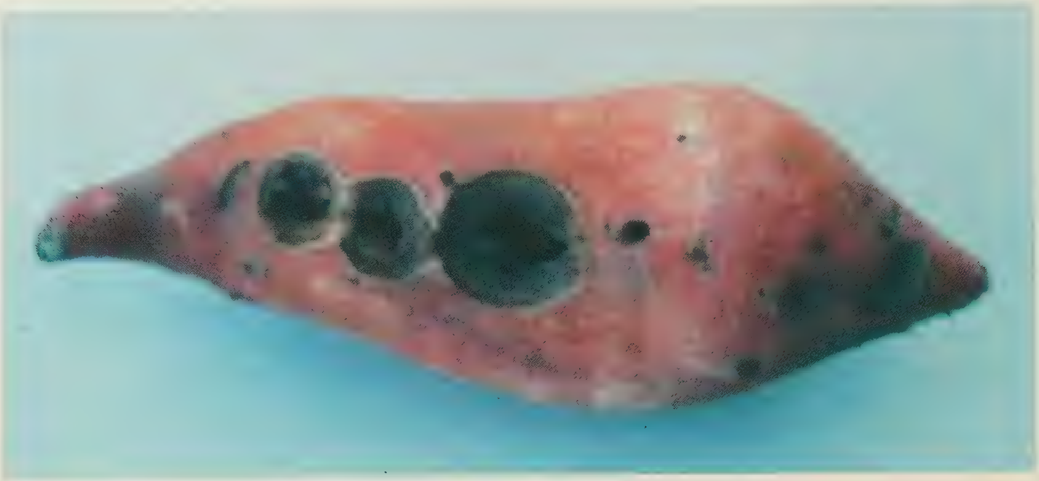
CONTROL Pieces of root used for seed should be dipped in a suitable fungicide before being planted (1). Harvested roots should be handled with care to minimise injury (10). They should not be left for too long in the field but removed to a shaded place in order to prevent excessive moisture loss. Immediate curing serves to heal wounds before the fungus can invade (5), and storage at 13° to 16°C is advantageous. When sweetpotatoes are brought out of store for washing and preparation for shipment and marketing, new injuries are inevitable. It has therefore been pointed out that re-curing is necessary, an appropriate strategy being to warm the roots in a hot water bath to enable them to cure subsequently whilst in transit (6). Cutting off the moribund tips of roots has been suggested as a means of reducing the likelihood of infection (9), and post-harvest fungicide treatments can be very effective (7,10). In winter care should be taken to protect sweetpotatoes from chilling injury (q.v.) since this predisposes them to rhizopus rot (6).

1 BOUWKAMP J.C., SCOTT L.E. & KANTZES J.G. (1971) Control of soft rot (*Rhizopus* sp.) in cut root pieces of sweetpotatoes with 2,6-dichloro-4-nitroaniline. *Plant Disease Reporter* **55,** 1097–1099.
2 DAINES R.H. (1942) Soft rot of sweet potatoes and its control. *Bulletin of New Jersey Agricultural Experiment Station* No. 698, 14 pp.
3 HARTER L.L., WEIMER J.L. & LAURITZEN J.I. (1921) The decay of sweet potatoes (*Ipomoea batatas*) produced by different species of *Rhizopus. Phytopathology* **11,** 279–284.
4 LAURITZEN J.I. & HARTER L.L. (1925) The influence of temperature on the infection and decay of sweet potatoes by different species of *Rhizopus. Journal of Agricultural Research* **30,** 793–810.
5 LAURITZEN J.I. & HARTER L.L. (1926) The relation of humidity to infection of the sweet potato by *Rhizopus. Journal of Agricultural Research* **33,** 527–539 illus.
6 McCLURE T.T. (1959) Rhizopus decay of sweetpotatoes as affected by chilling, recuring, and hydrowarming after storage. *Phytopathology* **49,** 359–361.
7 MARTIN W.J. (1964) Effectiveness of fungicides in reducing soft rot in washed, cured sweet potatoes. *Plant Disease Reporter* **48,** 606–607.
8 RANDLE W.M. & WOODSON W.R. (1986) The effect of storage and wounding on ethylene production by sweet potato *Ipomoea batatas. HortScience* **21,** 1018–1019.
9 SRIVASTAVA D.N. & WALKER J.C. (1959) Mechanisms of infection of sweet potato roots by *Rhizopus stolonifer. Phytopathology* **49,** 400–406.
10 WELCH N.C., PAULUS A.O. & SCHEUERMAN R.W. (1966) Rhizopus soft rot in sweet potatoes. *California Agriculture* **20**(11), 14–15 illus.

SCLEROTIUM ROT of sweetpotatoes caused by

Corticium rolfsii Curzi
Sclerotial state: *Sclerotium rolfsii* Sacc.

496



OCCURRENCE This disease has been recorded in the southern USA (2,4,5), Cuba (3), Jamaica, Israel, Mozambique (1) and India.

SYMPTOMS Various types of decay have been described. Lesions may be circular, approximately 1 cm in diameter, undergoing no further enlargement after harvest (4). On the other hand, rotting may be invasive, with a substantial amount of internal tissue becoming water-soaked yet firm, later hard and stringy (5). In a humid atmosphere there may be copious white mould growth, followed by production of small (1–2 mm) brown resting bodies (sclerotia) (page 255) (1,4).

BIOLOGY The sclerotia persist in the soil or on crop debris and, at the onset of warm wet weather, produce mould strands (1). The fungus requires good aeration, and tends to grow best in the upper soil layers, characteristically causing a 'collar rot' of the plant (2,3,5). Severe attacks can lead to wilting and death. Alternatively the fleshy roots may be invaded (3) and later decay in store. Post-harvest rotting is most severe under warm moist conditions (temperatures above 20°C) (5).

CONTROL Crop rotation is of little benefit in controlling this disease, since most crops are susceptible (3). Deep ploughing has been suggested as a means of burying sclerotia (5). Care should be taken to avoid contamination of new areas by inadvertent movement of infested soil on farm implements, animals etc. It may be necessary to fumigate the planting beds (4) and to improve drainage (1). It is especially important to use healthy planting material, since volatiles from decaying mother roots can predispose sweetpotato stems to collar rot (2). Plants which develop symptoms should be promptly removed and destroyed, together with the surrounding soil. Post-harvest decay can be held in check by storage temperatures of 10° to 12°C.

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2 CLARK C.A. (1989) Influence of volatiles from healthy and decaying sweet potato storage roots on sclerotial germination and hyphal growth of *Sclerotium rolfsii. Canadian Journal of Botany* **67,** 53–57.

3 GONZÁLEZ AVILA M. (1972) Algunos estudios sobre *Sclerotium rolfsii* en Cuba. *Revista de Agricultura, Cuba* **5**(2), 37–44 illus.

4 MARTIN W.J. (1953) Circular spot, a disease of sweet potato roots. *Phytopathology* **43,** 432–433 illus.

5 TAUBENHAUS J.J. (1919) Recent studies on *Sclerotium rolfsii* Sacc. *Journal of Agricultural Research* **18,** 127–138 illus.

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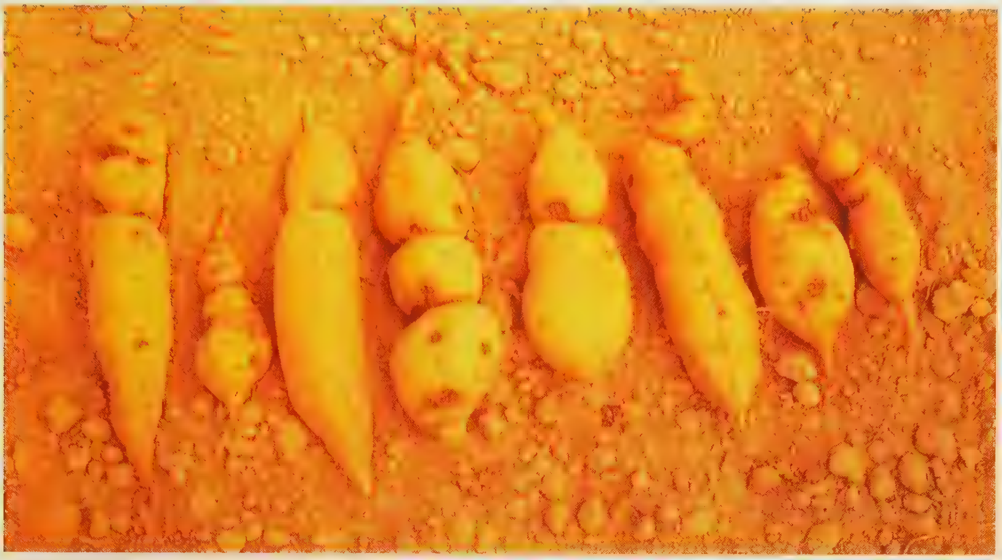
SOIL ROT or **POX** of sweetpotatoes caused by

Streptomyces ipomoeae **(Person & Martin) Waksman & Henrici**



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OCCURRENCE In some parts of the USA serious crop losses have resulted from this disease (4,5). It is caused by an actinomycete, an organism intermediate between a bacterium and a fungus (1,2). Lesions on harvested roots constitute an important market blemish (3).

SYMPTOMS Lesions are at first smooth and brown, but later the skin ruptures to expose conspicuous dark dry pits; they may be over 1 cm in diameter, coalescing to cover much of the root (4). A whitish growth indicates spore formation (1).

BIOLOGY The organism is soil-borne, and produces characteristic chains of diminutive spores (2). Dry weather and temperatures around 30°C favour infection, which typically occurs via the rootlets (1,4,5). If the root system is invaded early in the season, plant growth is stunted, the leaves turn pale and yields are reduced (3,4). Infected fleshy roots may be mis-shapen as well as blemished.

CONTROL Crop rotation is helpful, since other crops are not affected by this organism. Since a common means of spread is by infested soil, care should be taken to avoid contamination of new fields through movement of machinery, tools or livestock. When soil fumigation is impracticable, it has been found beneficial to add sulphur to infested soil, since soil rot does not develop in a very acid environment (pH 5.2 or below) (4). It may be advisable to dip planting material in a fungicide. Irrigation has been suggested as a simple means of control (5). New sweetpotato cultivars have been bred with resistance to soil rot, but they tend to be susceptible to other diseases such as sclerotium rot (q.v.). Breeding work continues (3).

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1 ADAMS J.F. (1929) An actinomycete the cause of soil rot or pox in sweet potatoes. *Phytopathology* **19,** 179–190 illus.

2 CLARK C.A. & LAWRENCE A. (1981) Morphology of spore-bearing structures in *Streptomyces ipomoea. Canadian Journal of Microbiology* **27,** 575–579 illus.

3 MOYER J.W., CAMPBELL C.L., ECHANDI E. & COLLINS W.W. (1984) Improved methodology for evaluating resistance in sweet potato to *Streptomyces ipomoea. Phytopathology* **74,** 494–497 illus.

4 PERSON L.H. & MARTIN W.J. (1940) Soil rot of sweet potatoes in Louisiana. *Phytopathology* **30,** 913–926 illus.

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See also:

CLARK C.A. & MATTHEWS S.W. (1987) Histopathology of sweet potato root infection by *Streptomyces ipomoea. Phytopathology* **77,** 1418–1423 illus.

SCURF of sweetpotatoes caused by
Monilochaetes infuscans **Ell. & Halst. ex Harter**

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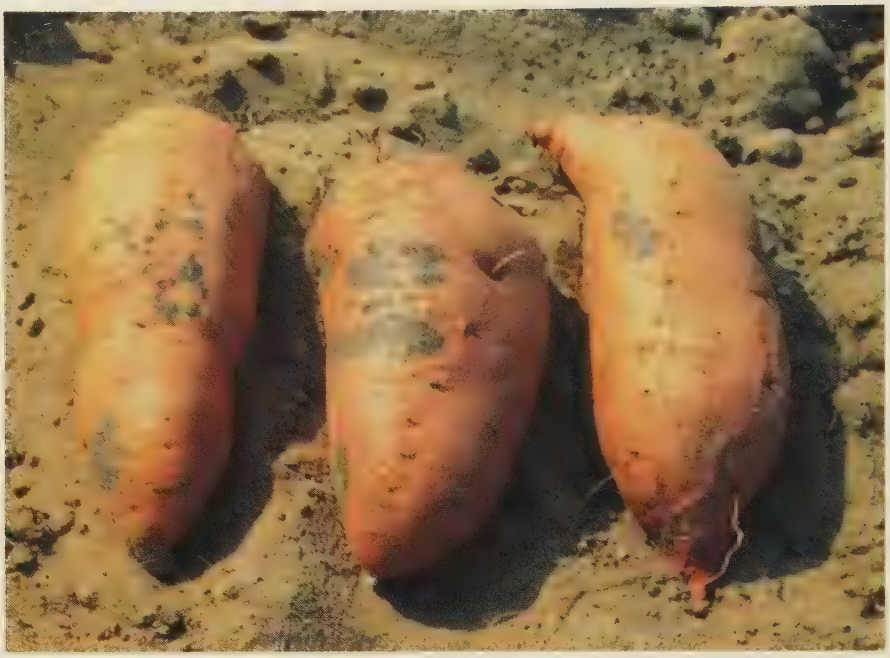

OCCURRENCE Countries in which the fungus has been recorded include Australia, New Zealand (7), Japan, China, Zimbabwe and Israel (CMI Map 246). The disease is common in the USA, and it is there that most of the the investigations have been carried out (1,2,3,4,5,8,9,10).

SYMPTOMS Small brown spots develop on the fleshy roots, later coalescing to form ill-defined irregular lesions (4,5). Characteristic dark spores (conidia) are produced (2,10). Although remaining superficial, scurf constitutes a blemish which detracts from market value (8,10). Furthermore, there is an increased tendency towards moisture loss and consequent shrivelling (2). Scurf-infected roots are also predisposed to invasion by other fungi (9).

BIOLOGY The fungus appears to be confined to sweetpotatoes and certain other species of *Ipomoea* (3). It persists in the soil on infected crop debris and, in wet conditions, germinating spores produce swellings (appressoria) which permit direct penetration of underground parts of the plant (4). The disease is spread by the use of contaminated planting material (1), and among the most susceptible cultivars are those possessing short attachment

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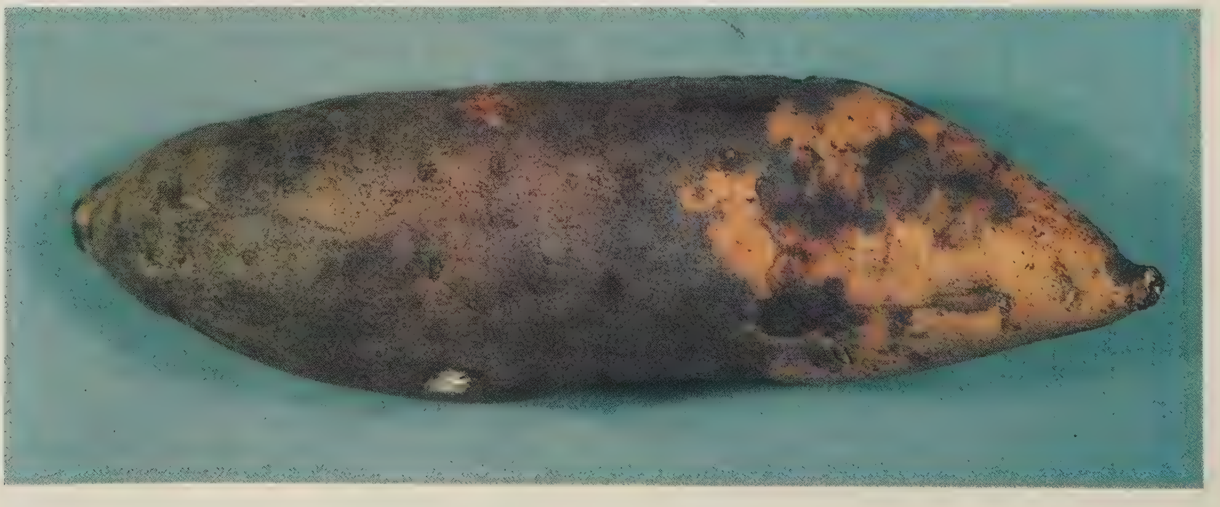

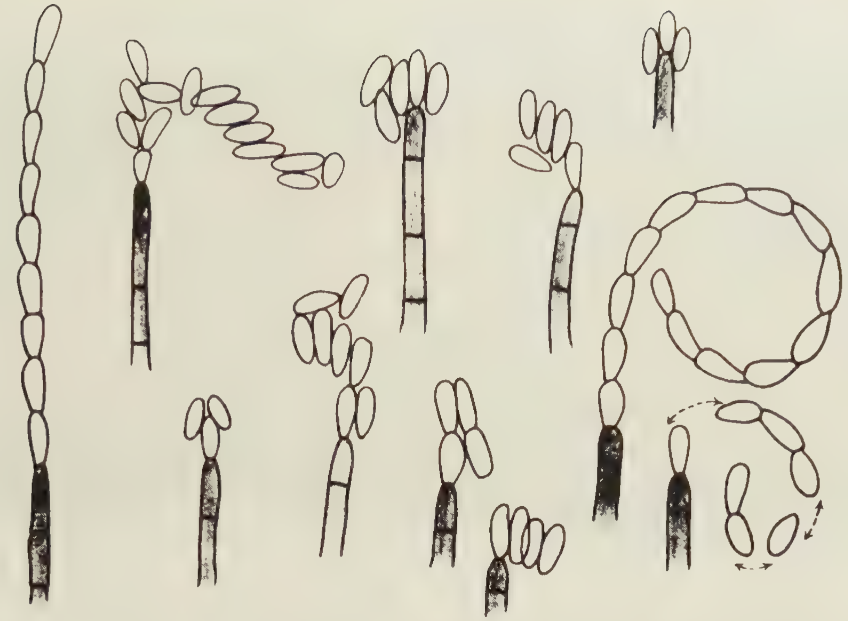

500 *Monilochaetes infuscans* (from *J. agric. Res.* **5**, 995–1002, Taubenhaus 1916) Conidiophores, each with chain of conidia, entire, breaking up after being disturbed or moistened, and bending prior to breaking up into individual spores

roots (the slender connections between mother root and daughter roots). The scurf fungus continues to develop in store and, if sufficient moisture is present, can also infect adjacent healthy roots (10).

CONTROL Harvested roots should be well-ventilated during storage (10). If roots are to be used for seed, they should be treated with an appropriate fungicide before planting (1,3), as careful selection alone is inadequate (8). Since the fungus does not infect above-ground parts of the plant (10), an effective control measure is to propagate sweetpotatoes by means of stem-cuttings (9), provided the soil is not infested. Crop rotation is beneficial (6), 2 to 3 years on light soils and 3 to 4 years on heavy soils. Although sweetpotato cultivars differ in reaction to infection there is apparently no high host resistance (7).

1 DAINES R.H. (1972) Effects of fungicide dips at various temperatures on the occurrence of sweetpotato scurf. *Plant Disease Reporter* **56,** 122–126 illus.

2 HARTER L.L. (1916) Sweet-potato scurf. *Journal of Agricultural Research* **5,** 787–792 illus.

3 KANTZES J.G. & COX C.E. (1958) Nutrition, pathogenicity, and control of *Monilochaetes infuscans* Ell. & Halst. ex Harter, the incitant of scurf of sweet potatoes. *Bulletin of Maryland Agricultural Experiment Station* No. A-95, 28 pp illus.

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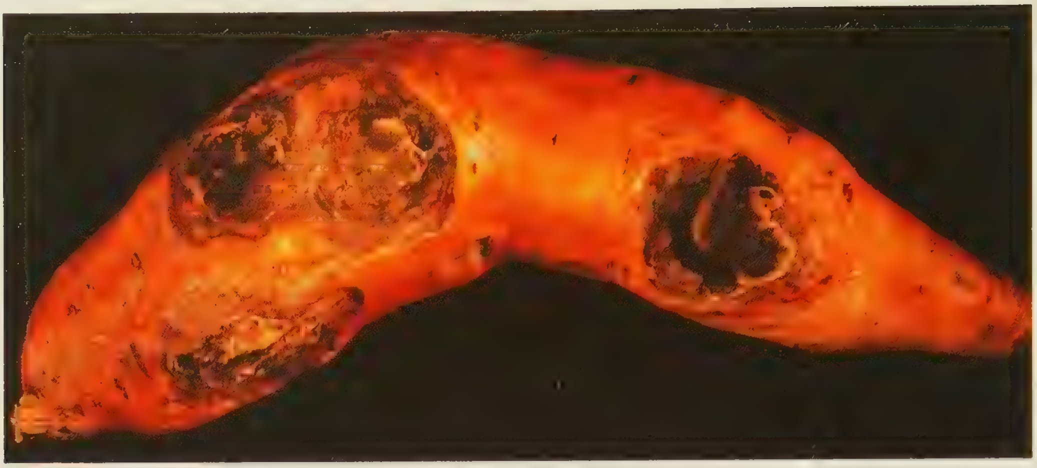
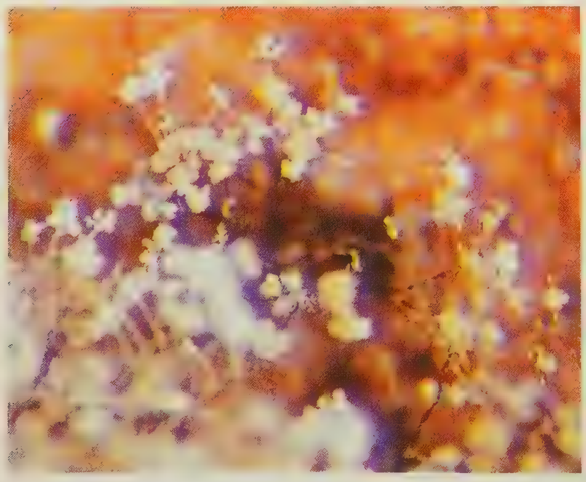
10 TAUBENHAUS J.J. (1916) Soil stain, or scurf, of the sweet potato. *Journal of Agricultural Research* **5,** 995–1002 illus.



501 Dry rot

BACTERIAL SOFT ROT, caused by *Erwinia chrysanthemi* (page 302), has been reported from the USA after exceptionally hot humid weather (9). Infected roots show black streaks in the vascular tissue, and subsequently undergo a soft moist decay. Rotting may occur in the field (where the first signs are wilting of the plant) and can also result in serious losses during shipment and storage. It may prove necessary to obtain clean seed roots from dry production areas where the disease is absent. Some sweetpotato cultivars are highly resistant.

BLUE MOULD ROT, GREY MOULD ROT and **MUCOR ROT** tend to occur only in roots which have been stored at low temperature and high humidity. Chilling injury (q.v.) predisposes the tissue to invasion by these weakly parasitic moulds, *Penicillium* spp., *Botrytis* spp. and *Mucor* spp., respectively.



502 *Botrytis cinerea*

CHARCOAL ROT is caused by the fungus *Macrophomina phaseolina* (page 25), which is widespread in the tropics. The disease is common in the southern USA and has also been reported from Egypt and Bangladesh. Decay of harvested roots usually begins at the point of original attachment to the plant, following 'collar rot' in the field. If an infected root is cut, three zones of affected tissue may be discerned – the advancing edge of the lesion is pale brown and spongy, the intermediate zone is reddish brown and firm, while the oldest part is almost black. The black colour appearance results from thousands of minute bodies (microsclerotia) which colonise the interior, but never the surface, of the root. Efficient curing of roots immediately after harvest is important for the rapid healing of injured tissue.

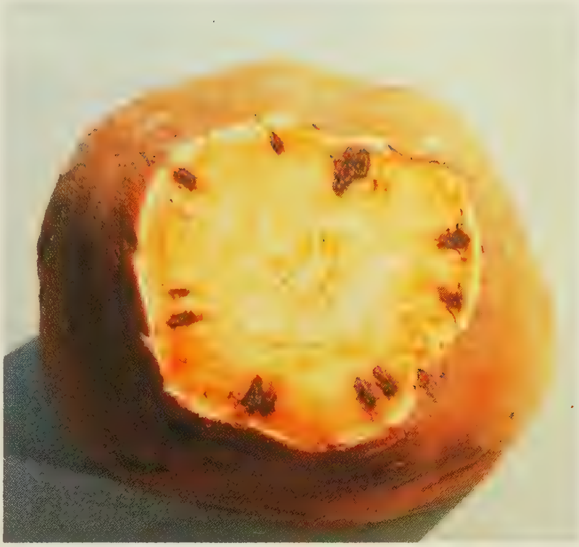
DRY ROT is caused by *Diaporthe phaseolorum* (Cooke & Ell.) Sacc. var. *batatatis* (Harter & Field) Wehm. (conidial state: *Phomopsis phaseoli* var. *batatatis*). It is widely distributed in the USA. Decay usually starts at one end of the root, infected tissue being dark brown and firm, becoming hard and shrunken as it loses moisture. Minute black bodies (pycnidia) develop in and beneath the skin. The fungus persists in sweetpotato debris, and can also be transmitted in infected roots used as seed. The chief means of control is to dip the roots in a fungicide (2).

FOOT ROT, caused by *Plenodomus destruens* Harter, is a serious field disease in parts of the USA (3). The fungus (which is now classified in *Phoma*) produces minute black bodies (pycnidia) which survive in crop debris. Spores are probably disseminated by wind, water, animals and equipment. Stems are attacked at soil level, and the fungus may advance into the fleshy roots. Further infections can occur via wounds sustained during harvesting and handling, and the organism may be transmitted in roots used as seed. Control involves limiting the transfer of infested material, sterilising seed beds and handling harvested roots with care (3).

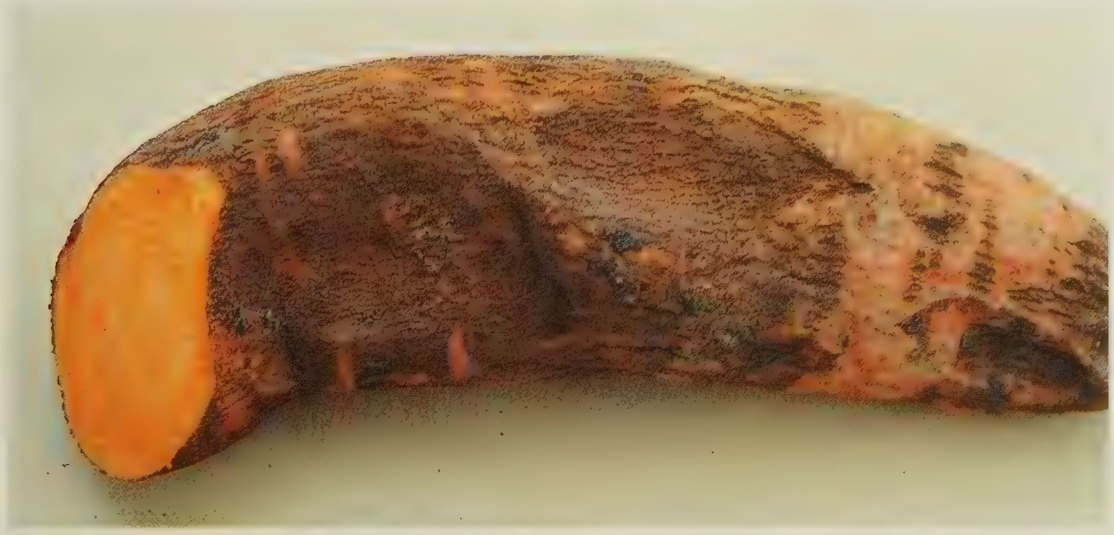
GLIOMASTIX ROT has caused losses in Egypt (5). The causal fungus is *Gliomastix novae-zelandiae*. Lesions appear as irregular brown corky areas, usually slightly depressed. The skin shrinks and is easily removed. In a humid atmosphere there is copious growth of black mould with abundant spores (conidia). The fungus is soil-borne and, since infection is via wounds, the chief means of control is efficient curing.

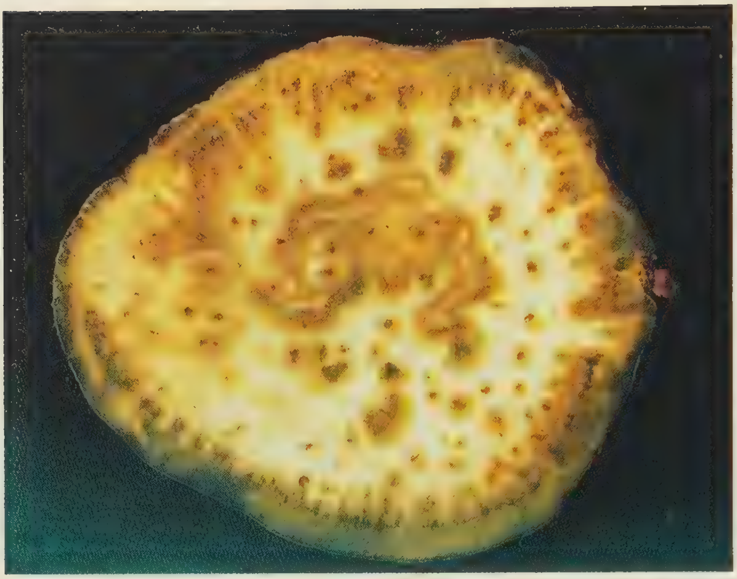
INTERNAL CORK is thought to be caused by a strain of the sweetpotato feathery mottle virus (SPFMV), which takes its name from the symptoms which may develop in infected leaves. Root symptoms consist of dark brown to black hard, corky lesions within the flesh (6). Small lesions are sometimes present at harvest, but extensive development occurs only after a period of storage at above-optimal temperature. Control involves the commercial production of healthy planting material, and also the breeding of tolerant sweetpotato cultivars (1,6).

PHYTOPHTHORA ROT, caused by *Phytophthora* sp., may take the form of **vascular browning** or **mottle necrosis**, as in pythium rot (q.v.).


503 Internal cork

PYTHIUM ROT can be serious after cool wet weather, and in the USA it is more common in northerly growing areas than in the hotter south. *Pythium ultimum* (page 328) appears to be the main causal organism; other species capable of inciting the disease include *P. aphanidermatum* (page 31), *P. scleroteichum* Drechsler and *P. splendens* Braun. A cross-section through freshly harvested roots may show irregular mottling or marbling, affected tissue being dry and crumbly, chocolate brown to dark grey in colour. The common name for this form of the disease is mottle necrosis (4). Alternatively, under different conditions, the decay extends uniformly through the root, and the flesh has a cheesy consistency. In a humid atmosphere white mould may develop. A third type of decay, designated ring rot, is characterised by a band of dark shrunken tissue which girdles the root, giving the appearance of a constriction (8). The chief means of control is early harvesting before the onset of cool wet weather.


504 Russet crack



505 Phytophthora rot

RHIZOCTONIA ROT has been reported from India (7), the causal fungus being *Rhizoctonia solani* (page 307). Pale brown spots develop, and affected roots tend to shrivel. Eventually the entire surface may be covered with a brownish mould, and large irregular-shaped black bodies (sclerotia) are produced, in a crust-like formation.

RUSSET CRACK describes a girdling of the root by corky brown lesions (1,6). As well as resulting in disfigurement, russet crack leads to increased moisture loss during storage. The cause is a strain of sweetpotato feathery mottle virus, different from the strain which causes internal cork (q.v.).

STEMPHYLIUM ROT is caused by the fungus *Pleospora herbarum* (page 211), and has been the subject of detailed study in South Africa (10). Lesions are at first soft, brown and moist, but as decay advances the tissues dry out and the root appears shrivelled. Internally the rotted area is bounded by a dark margin. Since infection occurs only through wounds, the main method of control is prompt curing of harvested roots.

WATERY SOFT ROT has been recorded on sweetpotatoes in transit in the USA. Decay may be caused by *Sclerotinia minor* or by *S. sclerotiorum* (page 47).

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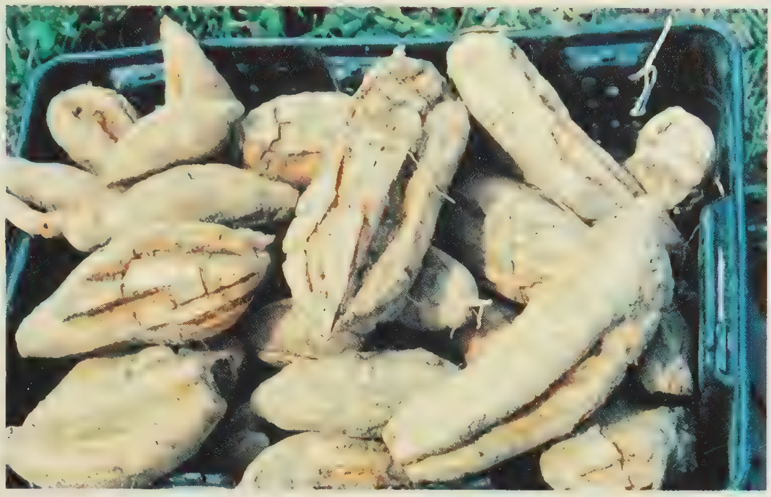
DISORDERS OF SWEETPOTATOES



506 Cracking caused by nematodes

CHILLING INJURY is the response to temperatures below about 13°C but above freezing point (5). The stress of low temperature exposure results in an increased rate of respiration together with irreversible physiological changes. In contrast to sound roots, chilled roots do not exude latex when cut. Other signs may include surface pitting, shrivelling, discoloration, and an increased tendency to decay (5). Some sweetpotato cultivars may appear normal in the raw state but fail to soften on cooking, this symptom being known as **hardcore** (1). Significant hardcore can result from exposure for one day at 1°C or for ten days at 10°C, followed by two or three days at higher temperature. In some growing areas sweetpotatoes may suffer chilling in the field, but the more usual cause of injury is a failure to protect consignments shipped to temperate destinations. Roots which have not been properly cured are less able to withstand low temperatures than are well-cured roots (1).

CRACKING may be caused by nematodes (eelworms) which are microscopic soil inhabitants (2,4). Cracks initiated early in the growing season are long and deep, while those induced at a later stage tend to be smaller but are more likely to be infected with secondary organisms. Shrinkage and decay ensue during storage. Control measures include crop rotation, chemical treatment of the soil, selection of nematode-free planting material, and the use of resistant cultivars (2,4).

INTERNAL BREAKDOWN can take the form of **pithiness** (3), in which parts of the flesh become white, dry and spongy, sometimes to the extent of developing cavities. Pithy roots are light in weight and lack firmness. The cause is excessive loss of moisture through evaporation and of dry matter through respiration. Predisposing factors include wounding, insufficient curing, and extended storage at above-optimal temperature and below-optimal humidity. Cultivars vary significantly in susceptibility.

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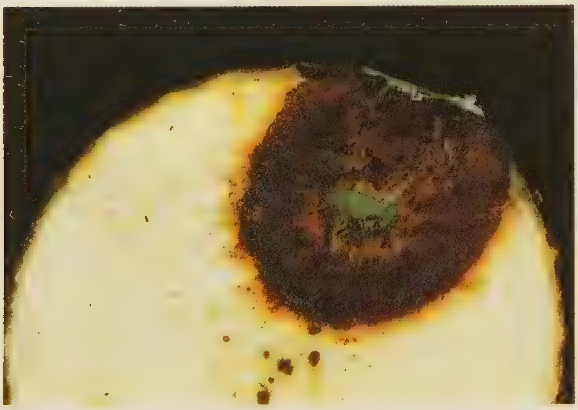
Yams

Yams are starchy tubers of the genus *Dioscorea,* which belongs to a family of monocotyledonous climbers (2,4,5,6). The ancestors of this family originated in the Far East; early in geological time they were distributed in the eastern and western hemispheres and various species evolved independently in each region.

The white yam, *D. rotundata* Poir, was first cultivated in West Africa, and was introduced into the West Indies in the 16th century. The tubers are large (30 to 40 cm in length) and generally cylindrical, with firm white flesh.



507 Green mould rot of white yam

Closely related is the yellow yam, *D. cayenensis* Lam., which is grown in Africa, the West Indies and parts of South America. Its tubers are of variable size and shape, and the flesh is usually pale yellow, although there are some cultivars which are almost white.

The greater yam, *D. alata* L., originated in South-east Asia and is now grown in nearly all parts of the tropics. The tubers are variable in size and shape, and have white or purplish flesh with a loose texture.

The lesser yam, *D. esculenta* (Lour.) Burk., has long been important in tropical Asia, and is now also grown in the Pacific Islands and the West Indies. It produces small (8 to 20 cm) cylindrical tubers with sweet white flesh.

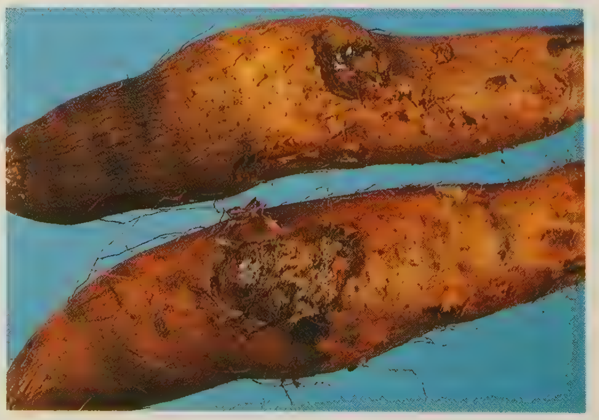
The cush-cush yam, *D. trifida* L., is grown in South America and the West Indies, and is the only yam of New World origin to attain significance as a food crop. It is eaten as a vegetable rather than as a staple, and has small sweet tubers which may be white, yellow, pink or purple.

There are many other types of yam, and considerable confusion exists both in common names and scientific names. In the USA sweetpotatoes (q.v.) are sometimes called yams.

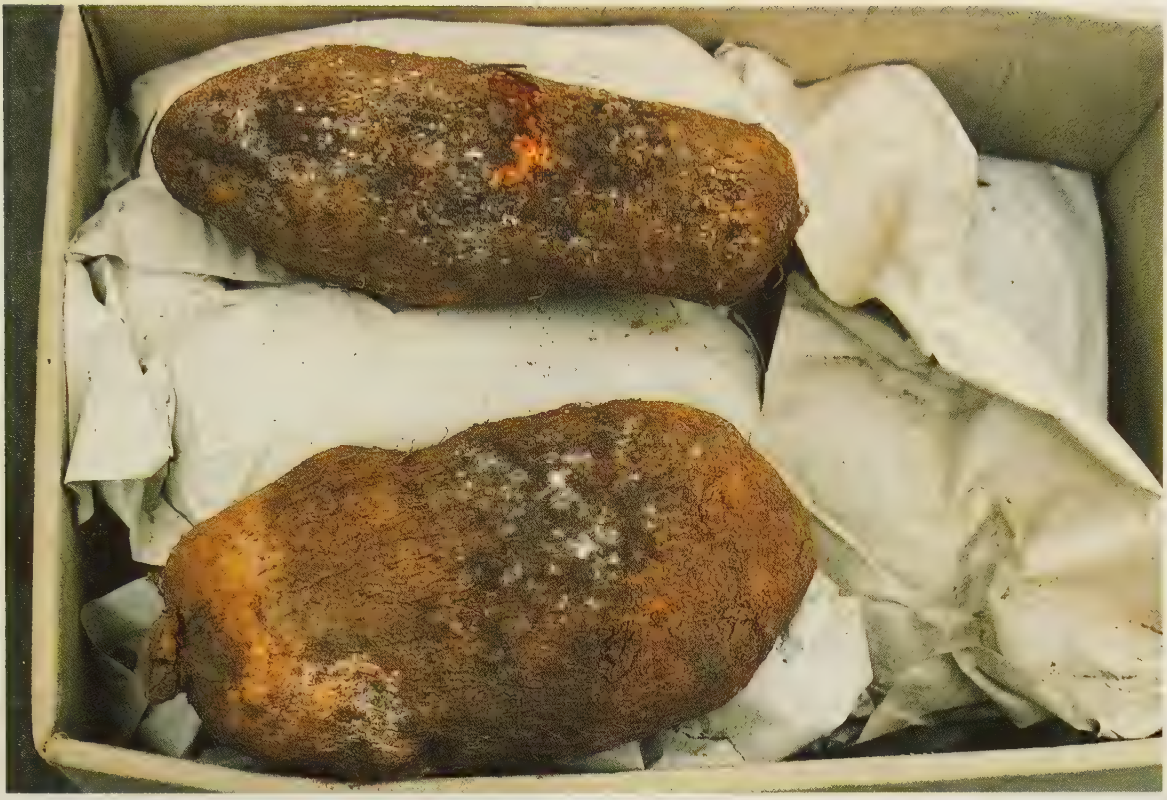
World production of yams is of the order of 27 million tonnes. By far the largest producer is Nigeria, where they are an important staple food. Other producing countries are the Ivory Coast, Ghana, Benin, Togo, Cameroon, Ethiopia, Zaire, Chad and Brazil. There is a substantial import trade in the UK, the main suppliers being Brazil, the West Indies and Ghana.

Yams are herbaceous plants with rampant twining stems, which are usually staked so as to ensure good leaf canopy formation. Under warm wet conditions each plant produces one or several tubers within a period of 7 to 9 months (2,6). Some tubers must be retained for cutting into seed-pieces. Depending on the climate, there may be two planting seasons per year. Furthermore, each of the two crops may be harvested either all at once (the tubers having reached full maturity) or twice (the tubers being carefully removed early in the season and the soil replaced, so that new tubers may develop). Control of eelworms (nematodes) is important, since infested tubers can later undergo serious deterioration during storage under tropical conditions (9).

It is difficult to harvest yams without causing injury. Consequently, if storage or shipment is intended, it is important to allow a period of curing immediately after harvest (1,2,3,6,8). Optimal conditions for wound-healing are a temperature of about 30°C and a relative humidity in the region of 95%. Such an environment may be achieved by lightly covering a pile of harvested yams with cut grass or jute sacks, taking care to avoid the risk of condensation. After 3 or 4 days the cured tubers can be removed, for storage in well-ventilated 'yam barns'. These vary in construction but, besides affording good aeration, they should provide shade from the sun and protection from rodents (2). Under such conditions yams may be kept for several months, the limiting factors being moisture loss (which results from respiratory losses as well as evaporation) (2), sprouting (10) and microbial decay (8). In some cultivars sprouting can



508 Cush-cush yams showing injury and decay



509 Chilling injury and mould damage

be inhibited by storing tubers in the dark or by using one of the growth-regulating chemicals (applied as a pre-harvest spray or a post-harvest dip) (10). As a post-harvest antimicrobial treatment, wood ash slurry can be as effective as expensive fungicides (7).

Deterioration is retarded by storage at moderate temperatures (9), but below 12° to 15°C yams are susceptible to chilling injury (q.v.) (8). Post-harvest fungicide dips should be used in conjunction with cool storage (3). The use of irradiation has been suggested as a cheaper alternative (3).

Yams for export should be carefully selected and packed in ventilated cartons or crates. Ventilated carriage is feasible; if a refrigerated ship is used, the carriage temperature should be about 15°C.

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ASPERGILLUS ROT of yams caused by
Aspergillus niger v. Tieghem

510 Conidial heads of *Aspergillus niger*

OCCURRENCE Aspergillus rot appears to be quite common, and has been recorded in Nigeria (1,3) and India (2), and also on yams imported into the UK from Jamaica (4,5).

SYMPTOMS Affected flesh is fawn or brown, sometimes tinged with purple. Lesions generally remain fairly firm, unless there is secondary infection by soft rot bacteria (1).

BIOLOGY The fungus thrives in tropical conditions, and its spores (conidia) are abundant in the atmosphere. Infection takes place via injuries sustained during harvest, handling or storage (3). Decay is especially rapid at temperatures between 25° and 35°C (1).

CONTROL Care should be taken to minimise physical damage (3), and a post-harvest fungicide dip may be beneficial (5). The fungus grows slowly at temperatures below 20°C.

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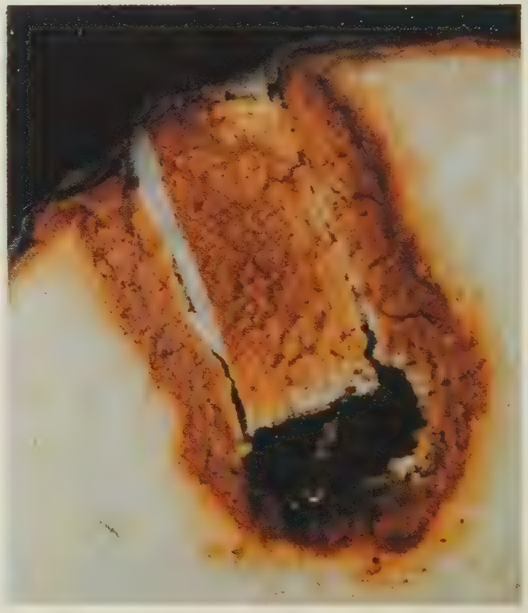

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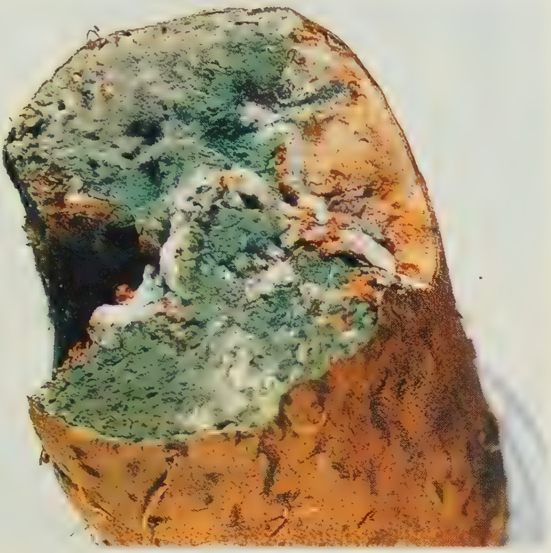
BLUE and **GREEN MOULD ROTS** of yams caused by
***Penicillium* spp.**

512



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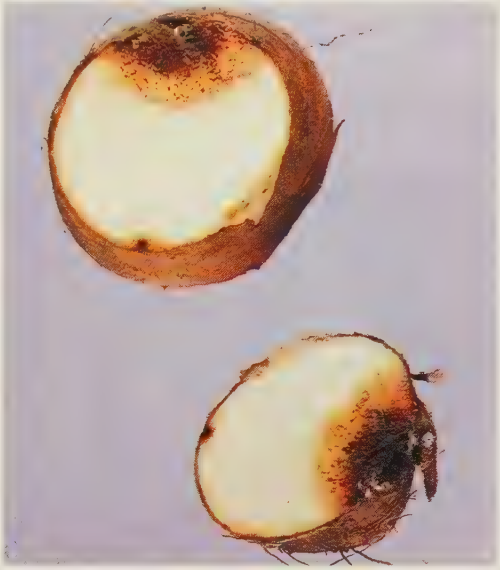

OCCURRENCE *P. crustosum* Thom is reported to be very common in yams imported into the UK from Nigeria, while *P. cyclopium* (page 244) is important in yams imported from Ghana. *P. gladioli* McCulloch & Thom has been found on importations to the USA from Cuba and Puerto Rico. *P. oxalicum* Currie & Thom has been recorded in Nigeria (1), Jamaica, and several islands of the French West Indies (4). *P. sclerotigenum* Yamam. was first described in Japan (5); subsequent records include India, Nigeria, Brazil (2) and Jamaica (3).

SYMPTOMS Blue or green mould growth is often associated with cut or damaged surfaces, but sometimes there may be a serious rot of the interior with no external symptoms. Rotted tissue is pale to dark brown, and may be firm or soft. Secondary bacterial infection can result in a wet rot. *P. sclerotigenum* produces minute brown spherical resting bodies (sclerotia) (5).

BIOLOGY These fungi survive in the soil on crop debris, and their spores (conidia) are dispersed by wind and water. Tubers are likely to be contaminated at harvest, and infection occurs via wounds (2), including the severed 'tail' of the tuber where it was originally attached to the plant (4). Decay by these species of *Penicillium* tends to be especially rapid at temperatures between 15° and 20°C (1).

CONTROL Tubers should be handled as carefully as possible so as to minimise injuries to the skin (4). Prompt use of a fungicide dip (within 3 days of harvest) can provide effective control of rotting in store (2,3,4). If fungicides are not available it is recommended that tubers be kept for several days in a warm humid atmosphere so as to promote rapid wound-healing (4).

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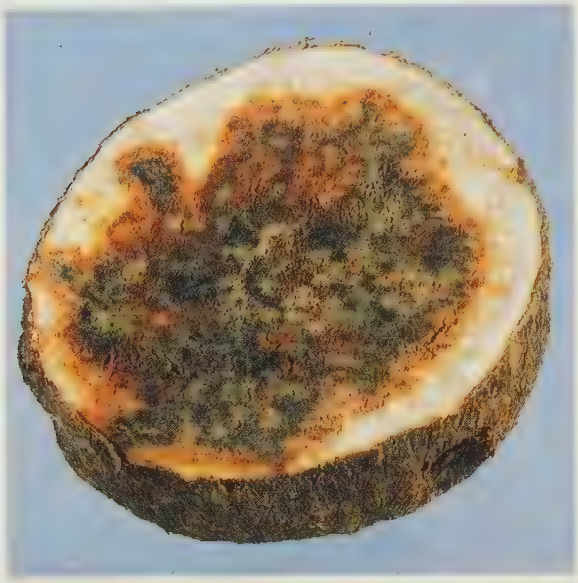
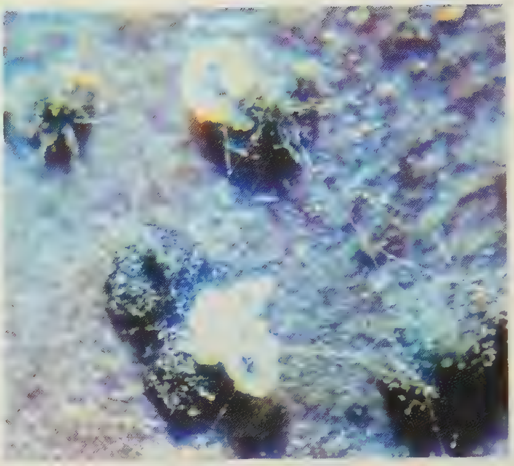
BOTRYODIPLODIA ROT of yams caused by
Botryodiplodia theobromae **Pat.**

514





515 Spore tendrils exuding from pycnidia

OCCURRENCE This disease has been reported from Ghana (3), the Ivory Coast, Nigeria (1,2,4) and India (5). It has also been recorded on yams imported into the UK from Ghana, Nigeria and the West Indies.

SYMPTOMS Affected tissue tends to be dark brown or black, and the margin between diseased and healthy tissue is usually a distinct brown line. Minute black bodies (pycnidia) eventually develop (page 371) (4). The nature of the rot may, however, be altered by the presence of secondary organisms which, together with environmental conditions, determine whether the rot is soft and water-soaked or firm and dry (3,4).

BIOLOGY The fungus survives for many months in crop debris and in soil, as thick-walled mould strands and as resting spores (chlamydospores) (2,4). In hot wet weather infection of tubers can occur in the field, via injuries made by farm implements or rodents (3). If harvested tubers are shipped to cooler countries, the temperature during transport and marketing is not usually high enough to allow extensive decay by this fungus. At 30°C, however, rotting is rapid (1,3) and the entire tuber may be invaded (4). After several months' storage, infected tubers become shrivelled 'mummies', in which may be found the asexual stage of the fungus (pycnidia giving rise to conidia). As the tubers crumble into flakes, pieces of infected tissue are dispersed by air currents (4).

CONTROL The most important measure is prevention of physical damage to the tubers. Care should be taken during cultivation of the crop, and animal pests should be controlled (3). Harvesting and handling should be as gentle as possible, and the use of a fungicide dip may be advisable. Since some physical injury is inevitable, a period of curing is necessary to permit wound-healing. At storage temperatures below 20°C, decay by this fungus is substantially reduced (1). If tubers are stored for long periods at high temperatures, inspections must be made and rotting tubers removed before they disintegrate (4).

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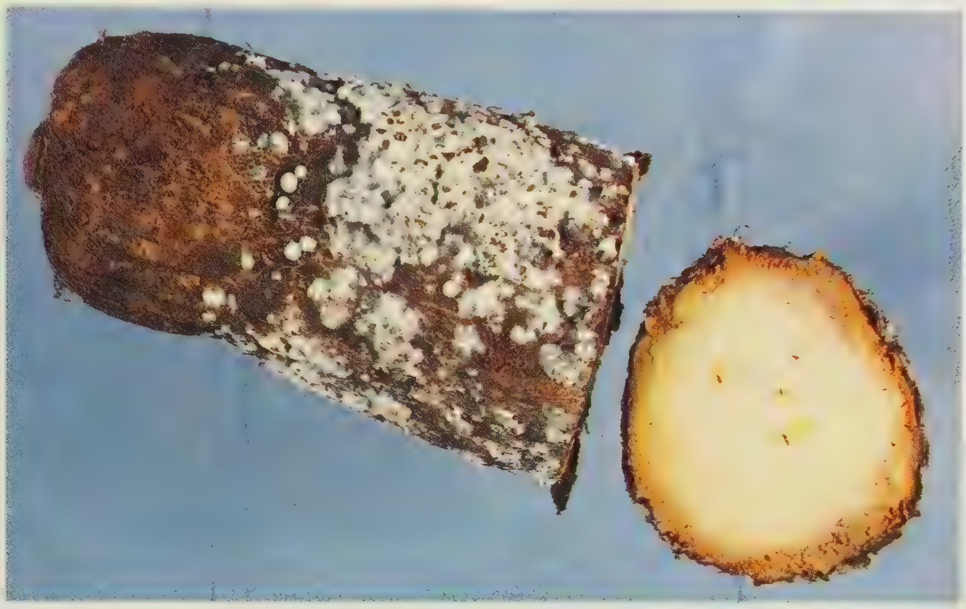
FUSARIUM ROT of yams caused by ***Fusarium*** **spp.**



516

OCCURRENCE *F. moniliforme* (page 353) has been recorded on yams in Nigeria (3,4), *F. oxysporum* (page 249) in Puerto Rico, and *F. solani* (page 369) in India (5). The latter two species have also been found on yams in or from Japan (1), Jamaica and Nigeria (2). The author recorded *F. oxysporum* following chilling injury in yams imported into the UK from Brazil.

SYMPTOMS Affected tissue is characteristically dry, pale in colour and bordered by a brown margin (1,2). In chill-injured tissue the texture is more likely to be soft and moist, and in a humid atmosphere the surface of the tuber becomes covered with tufts of dense white mould.

BIOLOGY These fungi are common soil inhabitants. It has been suggested that fusarium dry rot sometimes develops after invasion by eelworms (nematodes) which can attack yams either in the field or during storage. In any event, fungal spores are likely to be present on the surface of the tubers at harvest, and are capable of causing infection of tissue which has been injured mechanically or physiologically (2).

CONTROL It may be advisable to take control measures against nematodes (page 382). Harvested tubers should be handled with care and, if possible, dipped in a fungicide without delay (4). Additional protection is achieved by curing the tubers for several days at high temperature and high humidity to promote wound-healing (4), and subsequently storing them at a moderate temperature (page 390).

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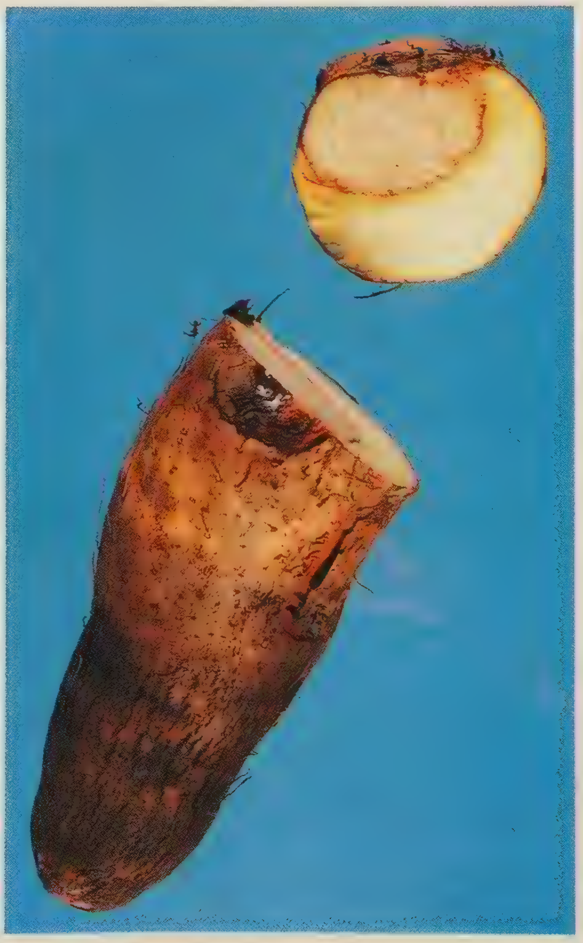
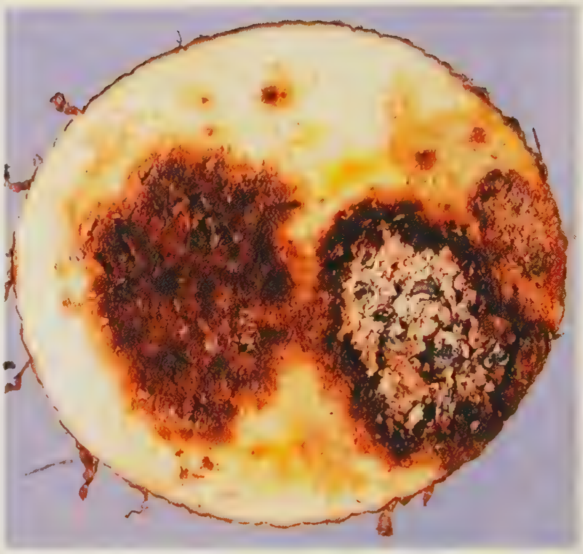
BACTERIAL SOFT ROT has been observed in yams imported from Puerto Rico into the USA, and also in yams shipped from Nigeria to the UK. The causal organism, *Erwinia* sp., has been described as a destructive secondary invader. Partial control can be achieved by the use of post-harvest treatment with hot water or heated wax. In Nigeria an orange-red pigmented bacterium, tentatively identified as *Serratia* sp., has been found to cause an extensive primary rot (9).

GREY MOULD ROT, caused by *Botrytis cinerea* (page 281), seems to occur only in yams weakened by chilling injury. It has been recorded on importations from Puerto Rico into the USA, and Jamaica into the UK, and the author has observed it in consignments shipped in refrigerated containers (at 8° to 10°C) from Brazil to the UK. Depending on conditions, infected tissue may be soft and wet or dry and crumbly.



517 Grey mould rot (wet)



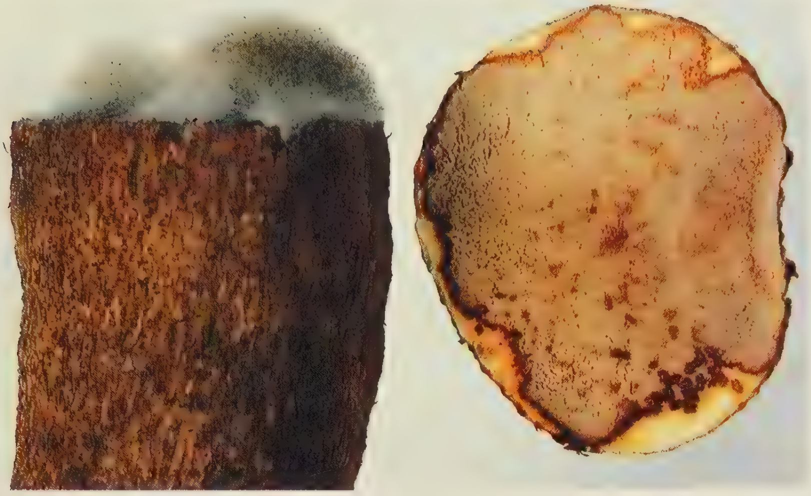
518 Grey mould rot (dry)

INTERNAL BROWN SPOT, thought to be caused by a virus, has been reported in several types of yam in Barbados (3) and neighbouring Caribbean islands (6). The symptoms, which are present at harvest, consist of small (0.5 to 2 mm) hard, brownish to black nodules in the flesh. Contrary to general belief, the spots do not increase in number during storage, although conditions of high humidity permit a slight increase in size and prominence. Tubers known to be infected should therefore be stored under well-ventilated dry conditions.

PYTHIUM ROT has been recorded on yams in Japan (4) and in India (10). *Pythium spinosum* causes a brownish decay in which there is a clear distinction between healthy and rotted tissue (10). Development is rapid at temperatures between 25° and 30°C, and in humid conditions a white mould forms on the surface. *P. sylvaticum* causes shallow, sunken, elliptical lesions, occasionally with lengthwise wrinkles; rotted tissue is soft and brown (4). *P. ultimum* (page 328) is said to cause brown lesions with a faint odour of violets. Yams which have been wounded, or weakened by chilling injury, are predisposed to infection by these fungi (4, 10).

RHIZOCTONIA ROT, caused by *Rhizoctonia solani* (page 307), has been reported from Japan and from India, as a decay of seed tubers (1). Fungicide treatment can be an effective control measure (1).

RHIZOPUS ROT, caused by *Rhizopus oryzae* (page 372) or *R. stolonifer* (page 81), has been recorded in



519 Rhizopus rot

Nigeria, the Ivory Coast (2), India (1), Brazil (7), and also on yams imported into the USA from Puerto Rico. It is sometimes known as 'watery rot' (7). Infected tissue is mottled brown and soft and, in a humid atmosphere, the cut surface is soon covered by a copious growth of coarse white mould strands. The characteristic spherical spore-heads are at first white and later black. Control measures include careful handling in order to minimise injury to the tubers. Application of fungicide in hot water or wax has been shown to give partial protection against invasion.

SCLEROTIUM ROT, caused by *Corticium rolfsii* (page 255), has been recorded in the Ivory Coast (2) and also on yams imported into Australia from China. In Nigeria this fungus causes a rot of yam seed pieces, and it is reported that curing in a shaded yam barn results in fewer rots than if pieces are cured on the soil surface shaded with palm fronds.

SPHAEROSTILBE ROT, caused by *Sphaerostilbe repens* (page 348), is typically a dry rot, and is reported to cause losses in the Ivory Coast (2).

YEASTY ROT, caused by *Candida* sp., is part of a disease complex reported from Nigeria (8). The fungus is capable of inciting a primary decay, but is usually associated with several other fungi such as *Aspergillus niger* and *Botryodiplodia theobromae* (q.v.). The resulting rot is soft and moist, with an odour of fermentation.

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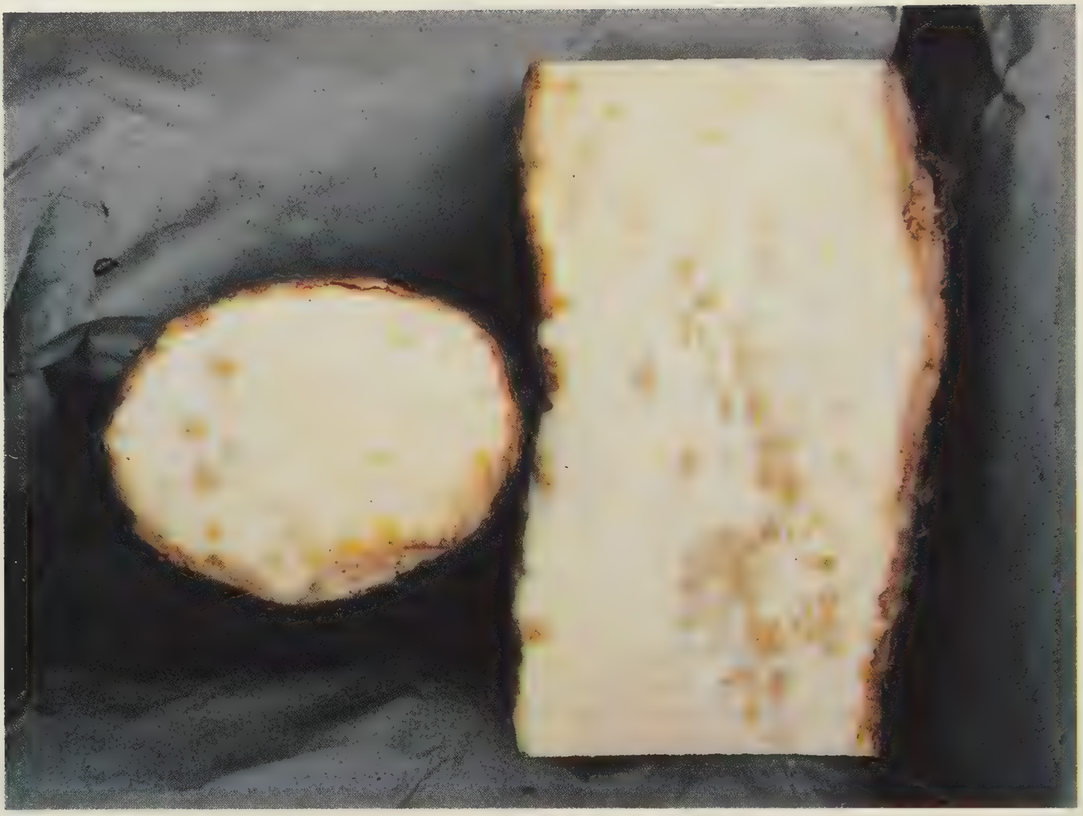
CHILLING INJURY of yams

520



OCCURRENCE Yams of all types may be injured by exposure to low temperature. Studies have been carried out on yams grown in, for example, the southern USA (5), Puerto Rico (1), Barbados (4), Jamaica (3) and Nigeria (2).

SYMPTOMS After slight chilling injury, yam tubers appear normal externally, but the flesh may be blotched (or, in longitudinal section, streaked) with yellow or brown (3). After severe injury the skin sloughs off easily, revealing a layer of soft discoloured tissue which is rapidly broken down by moulds (1). There is usually a substantial loss in weight (4).

BIOLOGY The critical temperature, below which chilling injury may occur, varies with species but can be as high as 10° to 12°C (2,3). Slight injury may be caused by 2 days at 4°C, or 5 to 10 days at 6° to 8°C followed by a short period at higher temperature for expression of symptoms (2). Chilled tissue is especially susceptible to grey mould rot (q.v.).

CONTROL If the optimal storage/carriage temperature is not known, it is suggested that yams be held at about 15°C. Precautions are needed when consignments are discharged in temperate countries during winter; delays should be avoided if the yams cannot be given protection.

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Appendix 1 Glossary of terms

(Origins of the technical terms are indicated in the Lexicon)

acaricide, a substance which kills mites.

acervulus, an erumpent, cushion-like mass of hyphae producing conidia on short conidiophores (Class Melanconiales).

aecium, a cup-shaped structure which produces aeciospores (Stage 1 in the rust fungi, Order Uredinales).

agar (from Malay **agar-agar**), a gelatine-like material obtained from seaweed and used to prepare culture media on which micro-organisms are grown and studied.

agronomy, the science of crop production.

alternate host, either of the two hosts of a heteroecious rust fungus.

alternative host, one of several plant species hosts of a given pathogen.

anamorph, asexual or imperfect state of a fungus.

antagonism, a general term for counteraction between organisms.

antheridium, the male sexual organ in some fungi (Class Oomycetes).

anthracnose, a plant disease having characteristic limited black lesions, usually sunken, with asexual spores in acervuli (Order Melanconiales).

antibiotic, a substance produced by a micro-organism and able to inhibit or kill other micro-organisms.

apothecium, a cup- or saucer-like structure containing asci and ascospores (Class Discomycetes).

appressorium, a thick-walled fungal cell formed prior to penetration.

Ascomycetes or, more strictly, Subdivision Ascomycotina, fungi which produce their spores in asci.

ascus, a sac-like structure containing ascospores (usually 8).

autoecious, (of rust fungus) able to complete its entire life cycle on one host species. cf. heteroecious.

bactericide, a substance which kills bacteria.

bacterium, a unicellular microscopic organism which lacks chlorophyll and which multiplies by splitting in two.

Basidiomycetes or, more strictly, Subdivision Basidiomycotina, fungi which produce their spores on basidia.

basidium, a club-shaped structure on which basidiospores are borne (Stage 4 in the rust fungi, Order Uredinales).

calyx, floral parts, consisting of sepals, remaining on some fruits such as apples, pomegranates, aubergines.

chilling injury, injury caused by low but non-freezing temperatures. cf. freezing injury.

chlamydospore, a hyphal cell, enveloped by a thick cell wall, which becomes separated from the parent hypha and behaves as a resting spore.

cleistothecium, a completely closed structure containing asci and ascospores (Class Plectomycetes).

climacteric, a period in the development of certain fruits, during which there is a pronounced increase in respiration, associated with the production of ethylene and leading to ripening.

clone, the aggregate of individual organisms produced asexually from one sexually produced individual.

conidiophore, a specialised hypha on which one or more conidia are borne.

conidium, an asexual spore which forms at the tip or side of a hypha.

controlled atmosphere (CA), an atmosphere in which oxygen, carbon dioxide and nitrogen concentrations are regulated, as well as temperature and humidity. cf. modified atmosphere.

cotyledon, the seed leaf of higher plants, one in the Monocotyledons and two in the Dicotyledons.

cultivar, a variety of a cultivated plant.

culture, growth of a micro-organism on artificial medium.

Deuteromycetes or, more strictly, Deuteromycotina, forms of fungi which produce asexual spores or no spores.

disease, harmful deviation from normal functioning of physiological processes, caused by one or more pathogenic organisms or viruses.

disorder, harmful deviation from normal functioning of physiological processes, arising from causes other than pathogenic organisms or viruses.

dormancy, state of minimal physiological activity.

downy mildews, diseases caused by fungi in the Family Peronosporaceae.

endoconidium, an asexual spore formed inside a hypha and extruded.

ethylene, a gas (C_2H_4) produced in small quantities by plant tissues.

exudate, liquid discharge from diseased or healthy plant tissue.

flagellum, a whip-like structure projecting from a bacterium or zoospore, serving to propel it through water.

freezing injury, injury caused by temperatures below the freezing point of the produce. cf. chilling injury.

fungicide, a substance which kills fungal spores or mycelium.

fungistat, a substance which prevents the growth of a fungus without killing it.

fungus, a type of organism which lacks chlorophyll and conductive tissues.

growth regulator, a natural substance which affects the enlargement, division, or activation of plant cells.

herbicide, weed-killer.

hermaphrodite, an individual bearing both functional male and female reproductive organs.

heteroecious, (of rust fungus) requiring two different host species for the completion of its life cycle. cf. autoecious.

horticultural maturity, the stage of development when a plant or plant part possesses the prerequisites for utilisation by consumers for a particular purpose.

host, an organism harbouring a parasite.

host range, the various kinds of host plants that may be attacked by a parasite.

hydathodes, structures with one or more openings that discharge water from the interior of the leaf to its surface.

hydro-cooling, cooling with cold water.

hypha, one of the threads of a mycelium.

immune, exempt from infection by a given pathogen.

imperfect fungus, a fungus which is not known to produce sexual spores.

imperfect state (anamorph), the state in which asexual spores (e.g. conidia) or no spores are produced.

infect, to enter and establish a permanent or temporary parasitic relationship with an organism.

infest, to over-run the surface of a plant, or to be dispersed through soil or other substrate.

inoculate, to introduce a micro-organism into an organism or into a culture medium.

isolate, to separate a micro-organism from its host or substrate and to establish it in pure culture.

lenticels, small pores in some plant organs (e.g. apple fruit, potato tuber), allowing the passage of water vapour and other gases.

lesion, a localised area of diseased or disordered tissue.

maturation, the stage of development leading to the attainment of physiological or horticultural maturity.

metabolism, the process by which cells or organisms utilise nutritive material to build living matter or to break down cellular material into simple substances to perform special functions.

modified atmosphere (MA), an atmosphere in which oxygen, carbon dioxide and nitrogen concentrations are different from those in air but are not precisely regulated. cf. controlled atmosphere.

mould (mold), superficial mycelium of a fungus.

mycelium, mass of fungal threads (hyphae).

mycology, the study of fungi.

mycotoxins, poisonous substances produced by certain fungi.

natural openings, stomata, lenticels, hydathodes.

nematicide, a substance which kills nematodes.

nematodes (eelworms), minute thread-like organisms living in soil, water or as parasites of plants and animals.

oogonium, the female sexual organ in some fungi (Class Oomycetes).

oospore, a sexual spore produced by the fusion of two morphologically dissimilar cells (oogonium and antheridium).

parasite, an organism living on or in, and getting its food from, its host, another living organism.

pathogen, a parasite able to cause disease in a particular host or range of hosts.

perfect state (teleomorph), the state of the life cycle in which spores (such as ascospores and basidiospores) are formed after nuclear fusion or by parthenogenesis.

perithecium, globose or flask-like structure (with an opening) containing asci and ascospores (Class Pyrenomycetes).

photosynthesis, the process by which carbon dioxide and water are combined in the presence of light and chlorophyll to form carbohydrate.

physiological maturity, the stage of development when a plant or plant part will continue to develop even if detached.

plant pathology (phytopathology), the study of plant disease.

plasmodium, a naked, slimy mass of protoplasm.

powdery mildews, diseases caused by fungi in the Family Erysiphaceae.

pre-cooling, removal of field heat prior to shipment.

pycnidium, globose or flask-like structure (with an opening) containing conidiophores and conidia (Order Sphaeropsidales).

pycnium (spermagonium), a flask-shaped structure containing pycniospores (spermatia) (Stage 0 in the rust fungi, Order Uredinales).

quarantine, (1) control of import and export of plants to prevent spread of diseases and pests; (2) holding of imported plants in isolation for a period to ensure their freedom from diseases and pests.

relative humidity (%), the ratio of the actual amount of water vapour in the air to the maximum it can hold at a given temperature, multiplied by 100.

resistant, (host plant) able to suppress or retard the activity of a pathogen; (micro-organism) able to withstand, completely or in some degree, the presence of a given chemical or other harmful factor.

respiration, a series of chemical oxidations within the cell, controlled and catalysed by enzymes, in which carbohydrate and fats are broken down, releasing energy to be used by the cell or organism in its various functions.

resting spore, a sexual or other thick-walled spore which is resistant to extremes of temperature and moisture and which tends to germinate only after a lapse of time.

ripening, the final stages of maturation when a fruit develops optimal appearance and eating quality.

rotting, the disintegration of tissues by the action of one or more micro-organisms.

rusts, diseases caused by the rust fungi (Order Uredinales).

sclerotium, a resting body of variable size, composed of a hard mass of hyphae with or without host tissue, usually with a darkened rind, from which may develop reproductive bodies, stromata, conidiophores or mycelium.

senescent, (tissue) growing old and dying.

sensitive, (host plant) reacting with severe symptoms to infection by a given pathogen; (micro-organism) succumbing to the effect of a given chemical or other harmful factor.

septate, possessing septa or cross-walls.

septum, a cross-wall in a hypha or spore.

seta, a bristle or stiff hair, generally thick-walled and dark coloured.

smuts, diseases caused by the smut fungi (Order Ustilaginales).

sooty moulds, sooty coatings on foliage and fruit, formed by the dark hyphae of fungi which live on the honeydew secreted by insects such as aphids, mealybugs and scales.

sorus, compact mass of spores, found especially in the rust and smut fungi.

spermagonium (pycnium), flask-shaped structure containing spermatia (pycniospores) (Stage 0 in rust fungi, Order Uredinales).

sporangiophore, a specialised hypha bearing one or more sporangia.

sporangium, a structure borne on a sporangiophore and containing sporangiospores (Class Oomycetes and Class Zygomycetes).

spore, the reproductive unit in fungi, consisting of one or more cells. It is analogous to the seed of green plants.

sporodochium, a cushion-like structure consisting of a mass of hyphae supporting conidiophores and conidia.

stoma, stomata, minute pores on the surfaces of leaves, stems and some fruits, allowing the passage of water vapour and other gases.

stroma, stromata, compact mycelial structures, with or without host tissue, in or on which spores are produced.

susceptible, subject to infection.

symptom, a detectable abnormality arising from disease.

syndrome, the totality of effects produced in a plant by a disease.

synergism, the concurrent parasitism of a host by two pathogens, in which the symptoms produced are of greater magnitude than the sum of the effects of each pathogen acting alone.

systemic, (of plant pathogen or chemical) occurring throughout the plant, having been translocated via the conductive system.

teleomorph, sexual or perfect state of a fungus.

teliospore, the sexual thick-walled resting spore of the rust and smut fungi.

telium, structure in which teliospores are produced (Stage 3 in the rust fungi, Order Uredinales).

tolerant, (host plant) able to endure infection by a particular pathogen, without showing severe symptoms; (micro-organism) able to withstand, completely or in some degree, the presence of a given chemical or other harmful factor.

transpiration, loss of water vapour from the surface of leaves and other plant organs.

uredinium, structure in which urediniospores are produced (Stage 2 in the rust fungi, Order Uredinales).

vacuum cooling, cooling by evaporation of water in a partial vacuum.

variety, (1) a subdivision below the level of sub-species; (2) a cultivar.

vascular, pertaining to the plant's conductive system.

vegetative, asexual.

vesicle, a bubble-like structure produced by a zoosporangium and in which the zoospores are released or are differentiated.

virulent, strongly pathogenic.

virus, a nucleoprotein entity able to pass through bacterium-retaining filters and able to replicate within the living cells of a host.

wilt, loss of rigidity and drooping of plant parts, generally caused by insufficient water in the plant or plant organ.

wound parasite, a parasitic organism which can invade a host only if it can first become established in damaged tissues.

yeasts, fungi which reproduce by budding.

zoosporangium, sporangium which contains or produces zoospores.

zoospore, spore possessing one or more flagella and capable of moving in water; a 'swimming' spore.

zygospore, sexual spore produced by the fusion of two morphologically similar cells (Class Zygomycetes).

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Appendix 2 Lexicon of Greek and Latin words
(from which are derived scientific names and technical terms)

If an organism has been well-named it is often possible to deduce some of its attributes. In the fungi, for example, *Albugo candida* is clearly *whiter* than *white, Phyllo/sticta* spp. cause *leaf spots,* while *Thanate/phorus cucumeris brings death* to *cucumbers* (and much else besides). *Cerato/cystis fimbriata* is characterised by the formation of a type of structure resembling a tough *horny bag* with a *fringe* (**484**). *Colleto/trichum circinans* possesses *sticky* spores (hence splash-dispersed rather than wind-borne), often interspersed with *hair-like* structures (setae); furthermore, the spore-masses tend to be arranged in concentric *circles* (**335, 336**). Less usefully, *Botryo/diplodia theo/bromae* is supposed to resemble in some aspects a *bunch of grapes,* its spores are (eventually) *2-celled,* and it lives on (amongst many other things) the *food* of the (Amerindian) *gods* i.e. cocoa, *Theobroma cacao* L. (**491**).

Technical terms (which are defined in the Glossary) tend to have Latin endings even though the original word may be Greek, for example **ascos** becomes **ascus**, **hyphe** becomes **hypha** and **basidion** becomes **basidium**. Latin plurals of words ending in **-us**, **-a** and **-um** are, respectively, **-i**, **-ae** and **-a**. A few technical terms remain pure Greek, for example **calyx** (plural **calyces**), **stoma** (plural **stomata**) and **stroma** (plural **stromata**). In the lists which follow, verbs are given in several forms and some nouns are quoted additionally in the genitive case where this is closer to the derived word. Greek transcription is largely based on Bodmer F. (1944) *The Loom of Language* (Ed. by L. Hogben), London: George Allen & Unwin.

GREEK

a-, an-	not, without
acares	too short to be cut
aecia	injury
agros	field
ampelos	vine
ana	up
angion	box, chest
antheros	flowering
anthos	flower
anthrax, anthracos	coal
anti	against, opposed to
apothece	storehouse
ascos	bag (wineskin)
aster (astron)	star
autos	self
bacter (bactron)	rod
basidion	small pedestal
bios, biotos	life
botane	herb, pasture
botrys	bunch of grapes
broma	food
bryone	moss
calyx, calycos	covering
carpos	fruit
cata	down
caulos	stalk
cephale	head
ceras, ceratos	horn
cercos	tail
chaete	long hair, mane
chalaros	slack, loose
chlamys, chlamydos	cloak
chloros	green, yellowish green
choane	funnel
chrysos	gold
cinnabari	vermilion
circinos	circle
clados	branch, sprout
cleistos	closed
climacter	rung of a ladder, critical stage
climax, climacos	ladder, ascent
clon	shoot
coccos	berry, grain
cochlias	snail
coelos	hollow
colla	glue
conis	dust
coryne	club
cotyle	cup
cryptos	hidden
cyclos	circle
cystis	bladder, bag
derma, dermatos	skin
deuteros	second, next
di	two
dia	among, through
didymos	twin
diploos	double

discos	dish, quoit, disc
dochion	receptacle
endon	inside
ergon	work
erythros	red
ge	earth
genete	birth
genos	kind, race, offspring
gloea	glue
gone	birth
gonos	offspring
helios	sun
helix, helicos	spiral, coil, snail
helmis, helminthos	worm
hermaphroditos	hermaphrodite (see Glossary) (the god Hermes and the goddess Aphrodite)
heteros	other, different
homos	same, similar
hydor, hydros	water
hyper	above, over, beyond
hyphe	web, tissue
hypo	beneath, under
idion	(diminutive)
idios	distinct
leptos	slender, delicate
leucos	white
logos	discourse, reasoning, word
lysis, lyticos	loosening
macros	long, large
melas, melanos	black
meli, melitos	honey
metabole	change
micros	small
monas	unit
monos	alone, single
morphe	form, shape
myces, mycetos	mushroom
myron	unguent, ointment
nema, nematos	thread
nomos	law, custom
oecos	house
oidion	little egg
oon	egg
opsis	appearance
oxys	sharp, acid
para	beside
pathos	suffering
peri	around
pezis	puff-ball
phaeos	dusky, grey
phagein	to eat, devour
pherein	to bear, bring, carry
phiale	bowl, saucer
philos	loving
phlyctaena	blister
phorein	to bear, bring, carry
phos, photos	light
phragma, phragmatos	fence
phthora	destruction
phycos	seaweed
phylaxis, phylactica	guarding
phyllon	leaf
phyma, phymatos	wart, pustule
physa	bellows
physis	nature
phyton	plant
plasma, plasmatos	form
plectos	plaited, twisted
pleon	more
pleos	full, infected with
polys	many
porthein	to destroy
pous, podos	foot
pseudos	false
pycnos	compact
pyren, pyrenos	fruit stone, pip
rhiza	root
rhodon	rose
scleros	hard
sitos	bread, food
soros	pile
sperma	seed
sphacelos	gangrene
sphaera	globe, sphere
sphongos	fungus, mushroom
spongia	sponge
sporos	seed
stasis, staticos	standstill
stemphylon	mass of pressed olives
stenos	narrow
stictos	spotted
stoma, stomatos	mouth
streptos	twisted, pliant
stroma, stromatos	mattress
syn	together, with
teleos	complete, perfect
telos	end
thanatos	death
thece	box, case, sheath
theos	god
thrips, thripos	wood-worm
thrix, trichos	hair
thyreos	shield
toxicon	poison
trachys	rough
ule	scar
xanthos	yellow
zoon	animal
zygon	yoke
zyme	yeast

LATIN

acarus	mite
acervulus	little heap
albus	white
alternus	one after the other
ante	before
apprimere, appressum	to press against
armilla	bracelet
artus	tight, close
asper, asperis	rough
aspergere	to sprinkle
aspergillum	holy water sprinkler
atramentum	black ink
bacillus (bacillum)	little rod
bi	two
caedere, cecidi	to kill
caeruleus	blue
candidus	shining white
canescens	becoming white
caput, capitis	head
charta	paper
cinis, cineris	ashes
cingula	girdle
clava	club
clavus	nail, spike
colo, colere	to inhabit
comosus	hairy
cortex, corticis	bark, rind, shell
creberrimus	crowded together
digitus	finger
exiguus	small, little, scanty
falcatus	sickle-shaped
fero, ferre	to bear, bring, carry
fimbriae (pl.)	fringe
flagellum	whip
flavus	golden yellow
forensis	relating to the law
fovea	pit
fuligo, fuliginis	soot
fulvus	tawny, yellowish brown
fumigatus	smoky
fungus	mushroom
funiculus	thin rope, cord, string
furfur, furfuris	scurf
fuscus	dark
fusus	spindle
galla	gall
gemma	bud
glomerella	little ball
herba	green plant, weed
hiemalis	of winter
humidus	wet, moist, damp
imperfectus	incomplete
inficere, infectum	to poison, taint
infuscere	to make dark, to blacken
latens, latentis	concealed, hidden
latericius	of brick
latex, laticis	fluid, liquid
laxus	loose, wide
lunatus	like the crescent moon
macula	spot
monile, monilis	necklace of beads
moribundus	dying
mors, mortis	death
necare	to kill
niger, nigra	black, dark
pallidus	pale
pannosus	ragged, tattered
penicillus	little brush
perfectus	complete, perfect
pirus	pear tree
planta	green twig, cutting
pomus	fruit tree
post	after
purpureus	purple, dark red
pustula	blister
quiescere	to rest, repose
racemus	cluster (especially of grapes)
ramulus	small branch
reticulum	little net
roseus	rose-coloured
scopae, scoparum (pl.)	broom made of twigs
senescere	to grow old
septum (saeptum)	wall, barrier, partition
seta (saeta)	bristle, stiff hair
squamus	scale
stria	furrow, groove
torulus	small swelling
trans	across, over, through
uncinula	little hook
uredo, uredinis	blight upon plants
uro, urere	to burn
ustulare	to burn, scorch
uva	bunch of grapes
vasculum	small vessel
vesicula	little bladder
viridis	green
virus	poison, venom
voro, vorare	to devour

Appendix 3 Recommended Storage Conditions

The conditions given are those which permit the longest possible duration of storage for the particular commodity, without undue loss of quality and allowing sufficient 'shelf-life' after the product has entered the marketing channels. If, however, a short period of storage is intended, it is often feasible to store produce at higher or lower temperatures and relative humidities than those recommended in the table.

Commodity Variety/Type/Origin	*Temperature* °C	°F	*Relative Humidity %*	*Storage Period*
Artichokes (Globe)	0 to 1	32 to 34	95–100	3–7 weeks
Asparagus	0 to 2	32 to 36	95–100	2–4 weeks
Aubergines	8 to 12	46 to 54	90–95	1–2 weeks
Beans				
Common	7 to 8	45 to 46	95–100	1–2 weeks
Faba	0 to 1	32 to 34	95–100	2–3 weeks
Beets				
Bunched	0 to 1	32 to 34	95–100	2–3 weeks
Topped	0 to 1	32 to 34	95–100	3–8 months
Broccoli	0 to 1	32 to 34	95–100	1–2 weeks
Brussels sprouts	0 to 1	32 to 34	95–100	3–5 weeks
Cabbages				
Green	0 to 1	32 to 34	95–100	3 months
White	0 to 1	32 to 34	95–100	6–7 months
Calabrese	0 to 1	32 to 34	95-100	1–2 weeks
Capsicums	7 to 10	45 to 50	90–95	1–3 weeks
Carrots				
Bunched	0 to 1	32 to 34	95–100	2–4 weeks
Topped (immature)	0 to 1	32 to 34	95–100	4–6 weeks
Topped (mature)	0 to 1	32 to 34	95–100	4–8 months
Cassava	0 to 2	32 to 36	85–90	5–6 months
Cauliflowers	0 to 1	32 to 34	95–100	2–4 weeks
Celeriac	0 to 1	32 to 34	95–100	3–5 months
Celery	0 to 1	32 to 34	95–100	1–3 months
Chayotes	9 to 11	48 to 52	85–90	4–6 weeks
Chicory	0 to 1	32 to 34	95–100	2–4 weeks
Chillies	7 to 10	45 to 50	90–95	1–3 weeks

Commodity *Variety/Type/Origin*	*Temperature* *°C*	 *°F*	*Relative* *Humidity %*	*Storage* *Period*
Chinese cabbages	0 to 1	32 to 34	95–100	1–2 months
Chives	0 to 1	32 to 34	95–100	1–2 weeks
Cocoyams				
('old' = taros)	11 to 13	52 to 55	85–90	5 months
('new' = tannias)	7 to 10	45 to 50	80	4–5 months
Courgettes	8 to 10	46 to 50	90–95	1–2 weeks
Cress	0 to 1	32 to 34	95–100	1–2 weeks
Cucumbers	8 to 11	45 to 52	90–95	1–2 weeks
Dasheen, Eddoe (Taros)	11 to 13	52 to 55	85–90	5 months
Eggplants	8 to 12	46 to 54	90–95	1–2 weeks
Endive	0 to 1	32 to 34	95–100	2–3 weeks
Fennel	0 to 1	32 to 34	95	1–2 weeks
Garlic	0	32	70	6–8 months
Ginger	13	55	70	4–6 months
Jerusalem artichokes	−0.5 to 0	31 to 32	90–95	2–5 months
Kale	0 to 1	32 to 34	95	2–4 weeks
Kohlrabi	0 to 1	32 to 34	95–100	2–4 weeks
Leeks	0 to 1	32 to 34	95–100	1–3 months
Lettuce	0 to 1	32 to 34	95–100	1–4 weeks
Mangetout	0 to 1	32 to 34	95–100	1–2 weeks
Marrows	10 to 12	50 to 54	60–70	2–6 months
Melons	5 to 10	41 to 50	90	1–4 weeks
Mint	0 to 1	32 to 34	95–100	2–4 weeks
Okra	7 to 10	45 to 50	95–100	1–2 weeks
Onions				
Green	0 to 1	32 to 34	95–100	1–3 weeks
Dry	−1 to 0	30 to 32	70–80	6–8 months
Parsley	0 to 1	32 to 34	95–100	1–2 months

Commodity *Variety/Type/Origin*	*Temperature* °C	 °F	*Relative Humidity %*	*Storage Period*
Parsnips	0 to 1	32 to 34	95–100	2–6 months
Peas	0 to 1	32 to 34	95–100	1–3 weeks
Peppers	7 to 10	45 to 50	90–95	1–3 weeks
Potatoes				
Immature	4 to 5	39 to 41	90–95	3–8 weeks
Mature	4 to 5	39 to 41	90–95	4–9 months
For processing into chips	7 to 8	45 to 46	90–95	6–9 months
For processing into crisps	8 to 9	46 to 48	90–95	6–9 months
Pumpkins	10 to 13	50 to 55	60–70	2–5 months
Radishes	0 to 1	32 to 34	95–100	1–4 weeks
Rhubarb	0 to 1	32 to 34	95–100	2–4 weeks
Rutabagas	0 to 1	32 to 34	95–100	4–6 months
Shallots	0 to 1	32 to 34	95–100	1–2 weeks
Spinach	0 to 1	32 to 34	95–100	1–2 weeks
Spring onions	0 to 1	32 to 34	95–100	1–3 weeks
Summer squashes	8 to 10	46 to 50	90–95	1–2 weeks
Swedes	0 to 1	32 to 34	95–100	4–6 months
Sweet corn	0 to 1	32 to 34	95–100	4–8 days
Sweetpotatoes	13 to 16	55 to 61	85–90	4–7 months
Tannias	7 to 10	45 to 50	80	4–5 months
Taros	11 to 13	52 to 55	85–90	5 months
Tomatoes				
Mature green	12 to 15	54 to 59	90	1–2 weeks
Turning	10 to 12	50 to 54	90	1–2 weeks
Ripe	8 to 10	46 to 50	90	1 week
Turnips	0 to 1	32 to 34	90–95	4–5 months
Watercress	0 to 1	32 to 34	95–100	3–4 days
Watermelons	10 to 12	50 to 54	90	2–3 weeks
Winter squashes	10 to 13	50 to 55	60–70	2–6 months
Zucchini	8 to 10	46 to 50	90–95	1–2 weeks

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- Cassava
- Cauliflowers
- Celery
- Chayotes
- Chicory
- Chinese leaves
- Chives
- Cucumbers
- Eggplants
- Endive
- Fennel
- Garlic
- Ginger
- Jerusalem artichokes
- Leeks
- Lettuce
- Marrows
- Melons
- Okra
- Onions
- Parsley
- Parsnips
- Peas
- Peppers
- Potatoes
- Pumpkins
- Radishes
- Rhubarb
- Shallots
- Spinach
- Spring onions
- Squashes
- Swedes
- Sweet corn
- Sweet potatoes
- Tannias
- Taros
- Tomatoes
- Turnips
- Watercress
- Watermelons
- Yams

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Volume 1 covers temperate and tropical fruits.

0-8493-7734-X

BC W7734